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THE PAINFUL SEQUELÆ OF INJURIES TO PERIPHERAL NERVES.[1]

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THE object of this paper is to report the results of an investigation of the painful sequelæ of injuries to peripheral nerves with special reference to causalgia.

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I. SOURCES OF PAIN AFTER INJURIES TO PERIPHERAL NERVES.

(1) The first extant record of pain after a gunshot nerve injury was furnished by Denmark,[1] whose patient was wounded in the arm at the storming of Badajoz in 1812. Though Denmark wrote of the radial nerve, it is clear from his context that his terminology was mixed and that it was the median that had been damaged. He relieved his patient by amputation at the mid-humeral level and examination of the arm revealed a pellet embedded in the nerve. Denmark was much impressed by the profound nutritive changes which occurred in the extremity of the damaged limb.

Because of the severe burning quality of the pain, which was such a feature in certain cases of nerve injury observed by him, Weir Mitchell[2] called it "causalgia". More recently some writers, following Stopford,[3] have suggested "thermalgia", on the grounds that the more familiar first syllable will convey its meaning to the reader at a glance. Mitchell, however, used the term merely to describe the main symptom and did not attach great importance to the word itself. To him, causalgia meant a continuous, severe, burning pain, of long duration, resulting from injury to the nerve. The syndrome was always seen in the limbs and nearly always in the hand or foot, which became moist, pink and glossy, while the patient was often a broken wreck whose chief concern was to protect the painful member from harmful stimuli.

"Causalgia" has achieved universal acceptance because it was coined by one of the masters of medicine and because it is a brief and expressive name for a little-understood syndrome. Though many writers on the subject describe it as a clear-cut "entity", in actual practice it presents many gradations in quality and intensity. At one boundary it shades off almost imperceptibly into some symptoms which are the usual effects of injuries to sensory nerves, and at another it is difficult to distinguish from manifestations which are uncommon effects of injuries to non-nervous tissues.

Boring[4] and Sharpey-Schafer,[5] who submitted themselves to section of a number of sensory nerves, felt burning, tingling pain in the denervated area during the first two weeks. Such sensations in a wounded soldier would be disregarded if they were not severe, and if severe and prolonged the pain would be called "causalgia". It is possible, therefore, that "causalgia" represents the prolongation and intensification of a common effect of injury to nerves.

(2) The site of the injury to the nerve may be occupied by a tender neuroma, pressure on which causes pain or other unpleasant sensations to radiate to the extremity. The pain, however, is not spontaneous and is not related to causalgia.

(3) Kellgren,[6] Fay,[7] Lewis,[8] Inman and Saunders[9] and others have observed, from experimental lesions of muscular, fibrous and vascular tissues situated in the proximal parts of a limb, that pain may radiate to the hand or foot. When a nerve has been injured, non-nervous tissues have been damaged also and this damage may be prolonged by infection. In such a case, every pain felt in the extremity is not necessarily the result of the nerve injury.

(4) Spreading pain, with atrophy of the tissues, sometimes follows a minor injury to the hand or foot; the condition has been described by a number of writers under different names: ascending neuritis;[3] Sudeck's atrophy;[10] post-traumatic painful osteoporosis (Miller and de Takats[11]); post-traumatic spreading neuralgia (Leriche[12]); minor causalgia (Homans,[13] Livingston[14]); "causalgic states" (de Takats[15]). Of Rasmussen and Freedman's[16] 100 cases of causalgia, a nerve trunk was injured in 85; in the remainder the pain followed injury to bone and other structures.

(5) Somatic pain is sometimes associated with cutaneous hyperalgesia in which a lesion of a nerve trunk has been assumed but not proven. Thus Hudson, Hettesheimer and Robin,[17] having observed hyperalgesia to accompany backache, presumed that a lumbar nerve must have been nipped as it emerged from the spinal column; they described the disorder as "causalgic backache". Magee[18] wrote of "genito-femoral causalgia" when hyperalgesia was detected in association with a painful appendicectomy scar. But the presence of hyperalgesia is not pathognomonic of interference with a nerve trunk; it is well known to occur with visceral disease. Lewis and Kellgren[19] frequently observed this sign to accompany the pain of experimental somatic lesions. In these two instances, though the pain has been described as "causalgic" (that is, burning), an essential feature of the syndrome (proven injury to a nerve trunk) is missing.

(6) Painful amputation stumps and phantom limb pain. Pains of different types may be included in this class, but, according to Livingston,[20] the commonest is "a burning sense of heat, as if the hand were held too close to a fire". It is likely that the pain in these cases has resulted from the nerve injury, but because of the many additional structures involved they were excluded from the present study.

(7) When a limb has been encased in plaster for several weeks, it is difficult to say whether or not pain is due to pressure or to an uncomfortable position.

(8) During the process of recovery aching may occur in the injured limb through over-use of the weakened muscles. Many patients who are recovering from sciatic nerve lesions complain of pain after walking. Deep tenderness may be observed for a time in muscles which are recovering from paralysis.

(9) Many complain of aching pain if the denervated parts become cold. This is but an accentuation of the usual painful response to cold.

(10) During recovery of sensation the skin may be the site of hyperalgesia which, in many causalgic cases, may be intense. Often, however, exquisite hyperalgesia is observed without any history of spontaneous pain.

II. CLINICAL MATERIAL AND THE SELECTION OF CASES.

During and after the recent war one of us (S.S.) had under his care 339 lesions of individual peripheral nerves in 301 patients. For the purpose of this report only primary nerve lesions have been included, that is, those which followed immediately after the injury. Such lesions as cervical rib and late ulnar palsy have been excluded, as have all lesions of the posterior interosseous nerve because of the absence of cutaneous fibres. The number of patients is therefore reduced to 278 and of individual nerves to 312. There were four patients in whom the same nerve was injured on both sides. For the purposes of this paper these bilateral lesions have been treated as individual cases—the number of patients thus becomes 282 (see Table I).

The majority of the patients arrived at the clinic several weeks after sustaining the injury, and it was not always easy to secure an accurate history of the patient's sensations during the first few weeks. Often he had been very ill, with multiple wounds, severe infection or a grossly comminuted fracture, and it was only when the skeletal injuries were being brought under control that attention was directed to his nerve pains. It should not be surprising, therefore, if the early records are sketchy or if the patient cannot give a clear account of his early sufferings.

Several writers have not attempted to distinguish the painful sequelæ of injuries to nerves from those which follow damage or stress to other

tissues. The purpose of the present review, however, is to deal only with the effects of injury to nerve trunks, and no case has been included for study which did not conform to the following features: (i) The pain followed an injury to a large nerve trunk. (ii) It was severe. It has not been thought necessary to insist that the pain should have a burning quality, for in nearly half of the severest cases in the present study it was described in other terms (for example, crushing, tearing, "like a severe toothache" *et cetera*). (iii) It was felt in the hand or foot. (iv) Its duration was longer than five weeks. (v) The pain was not related to damage to or involvement of non-neural tissues.

When the doubtful cases had been excluded there remained 34 cases in which severe pain in the extremity had persisted for at least five weeks after the receipt of an injury to a nerve trunk, and the term "causalgia" has been applied to this pain. In the majority of these cases, thanks to the generous

TABLE I.

Incidence of Causalgia in Nerve Lesions.

	Number of Cases.	Causalgia.
Sciatic:		
Gunshot wound *plus* bone injury	15	5
Gunshot wound	29	10
Closed fracture (or dislocation)	5	1
Total	49	16
Medial popliteal:		
Gunshot wound *plus* bone injury	1	—
Gunshot wound	2	—
Total	3	—
Lateral popliteal:		
Gunshot wound	6	1
Other causes	9	—
Total	15	1
Other nerves in lower limb	3	—
Total	3	—
Single lesions upper limb:		
Brachial plexus:		
Gunshot wound *plus* bone injury	3	1
Gunshot wound	12	3
Stretch (with or without fractured clavicle)	10	1
Total	25	5
Median single:		
Gunshot wound *plus* bone injury	9	1
Gunshot wound	18	5
Miscellaneous	5	—
Lacerations	10	—
Total	42	6
Radial:		
Gunshot wound *plus* bone injury	20	—
Gunshot wound	11	—
Closed fracture	4	2
Lacerations	4	—
Compression	7	—
Total	46	2
Ulnar:		
Gunshot wound *plus* bone injury	15	—
Gunshot wound	22	1
Lacerations	12	—
Other causes	15	—
Total	64	1
Other nerves	2	—
Total	2	—

TABLE I.—*Continued.*

Incidence of Causalgia in Nerve Lesions.

	Number of Cases.	Causalgia.
Combined lesions upper limb:		
Radial-median-ulnar:		
Gunshot wound *plus* bone injury	1	—
Gunshot wound	1	—
Tourniquet compression (projectile responsible for most of median component)	2	1
Total	4	1
Radial-median:		
Gunshot wound *plus* bone injury	2	—
Gunshot wound	1	—
Closed fracture	1	—
Lacerations	1	—
Total	5	—
Radial-ulnar:		
Gunshot wound *plus* bone injury	6	—
Gunshot wound	1	—
Simple compression	1	—
Total	8	—
Median ulnar:		
Gunshot wound *plus* bone injury	2	—
Gunshot wound	9	2
Lacerations	3	—
Miscellaneous	1	—
Total	15	2
Median-posterior interosseous:		
Gunshot wound *plus* bone injury	1	—
Total	1	—
Grand total	282	34

cooperation of the Repatriation Department, it has been possible to maintain personal interviews with the patients over periods of two to five years after the injury.

The proportion of causalgic cases to the total number of nerve injuries is higher than in the series reported by Rasmussen and Freedman,[16] Lewis and Gatewood,[20] Carter,[21] Speigel and Milowsky,[22] Mayfield and Devine,[23] Ulmer and Mayfield,[24] and Kirklin *et alii*.[25] The observations, however, were made at a base hospital in Australia to which patients with severe nerve injuries and those with intractable pain were referred. Patients with mild lesions recovering rapidly with acute but only transient pain would not, in most instances, reach such a centre. Lewis and Gatewood held that only the intense paroxysmal type should be regarded as causalgia; only four cases out of their total of 550 nerve lesions were thought severe enough to merit the description. The criteria accepted by Rasmussen and Freedman were less rigid and pains of four different grades of severity were included in their series. The standards demanded for the use of the term necessarily depend upon the views of each observer. When, however, the symptoms vary only in severity, there is no reason for believing that the mechanism of pain production is different; valuable lessons can be learned from some of the cases of milder degree.

III. CASE RECORDS.

In the records which follow, those details only have been mentioned which are considered relevant to the present discussion and, for brevity, negative observations have been omitted. Difficulty has proceeded from the fact that "causalgic" pain usually occurs in the first few weeks after injury,

when the wounded soldier is being transferred from hospital to hospital and coming under the care of a succession of medical officers, none of whom is able to make prolonged observations. The clinical notes made during this period are often brief and hurried and, on questioning the patient at a long interval afterwards, one cannot be sure that his memory is accurate for all details.

Sciatic Nerve.

CASE 1.—W.G. sustained a perforating bullet wound of the upper part of the left thigh, which resulted in a subtrochanteric fracture and a complete sciatic palsy. At 35 weeks there was a flicker of contraction in all muscles supplied by the sciatic nerve; as this condition remained stationary the damaged section of the nerve was explored at 51 weeks and found to be in continuity, with epineural and intraneural fibrosis. Electrical stimulation of the nerve resulted in the contraction of all muscles. Neurolysis was performed. Motor function commenced to improve after this operation and, three years after the injury, was normal. The field of sensory loss was difficult to determine accurately from the first examination (at 35 weeks); the sole appeared normal, while sensation to pin-prick was defective over the lateral popliteal field. Hyperalgesia was never recorded and within two years sensation was normal.

Pain.—A few minutes after the patient was hit severe pain commenced in the heel. Subsequently he complained of continuous severe aching pain, which was centred in the heel, but which spread through the foot from this source; sometimes lancinating pains shot into the toes. One hundred and three days after the injury the records stated "under spinal 6 c.c. percaine-injection 2½ c.c. proctocaine into sciatic nerve; anæsthesia of the foot following this injection with relief of pain for 18 hours". Thirty-eight days later the nerve was exposed and injected with 5·0 cubic centimetres of "Proctocaine". This procedure afforded only temporary relief (18 to 48 hours). The pain did not become less after the neurolysis, 51 weeks after injury, and a lumbar sympathectomy was performed five weeks later. The pain was relieved temporarily, but at 129 weeks he was complaining of a severe pain in the foot, mostly in the heel, which was worse at night. At 264 weeks he still had pain in the heel, shooting forward to the toes, with sometimes a sensation as though "wires were going through the foot"; this pain was worse at night and when the foot became warm by the fire.

CASE 2.—J.L.G. sustained a perforating bullet wound of the upper part of the left thigh, which resulted in a fracture of the femur and paralysis of all the muscles below the hamstrings, with complete anæsthesia in the medial and lateral popliteal fields. The soft tissues were severely damaged and infection followed; trophic changes occurred rapidly with wasting and intractable ulceration of the sole. Within 13 weeks all the muscles except the flexors and extensors of the toes were contracting; extension of the toes was achieved at 21 weeks and proceeded to good recovery, but after two and a half years there was only a flicker of contraction in the flexors. Early anæsthesia soon changed to exquisite hyperalgesia; at two and a half years there was a large area of grossly diminished sensibility on the sole and outer surface of the lower leg.

Pain.—A week after the patient was wounded a severe stabbing pain commenced in the foot; sometimes it would travel up the leg, but it was mostly centred in the foot. Any knock or jerk on the foot would precipitate an attack. It was made worse by cold and lessened by warmth. Lumbar sympathectomy was performed at 32 weeks; the pain was not relieved immediately, but commenced to become less a few days after the operation. At two years he was getting mild attacks of pain in bed on three nights out of four. If he walked 300 yards he would feel "as though a red-hot knife was being driven into the foot". At two and a quarter years the causalgia was no longer troublesome.

CASE 3.—L.R. was wounded (penetrating shrapnel) in the upper part of the left thigh and sustained a complete sciatic paralysis. When the nerve was exposed at operation three weeks later its appearance was "normal". Motor recovery in the lateral popliteal division was rapid and slower in the medial division; at 38 days all the muscles except the long flexors of the toes were contracting. At 19 weeks the motor field of the lateral popliteal had recovered completely, while there was only slight activity in the medial popliteal field. Seventeen days after the injury the lateral popliteal field was hyperalgesic and the medial hypoalgesic; at nine weeks cutaneous sensation was normal. The calf muscles were very tender to pressure, while those in the anterior compartment gave a normal response to pressure.

Pain.—A week after the injury the patient was complaining of continuous severe pain in the sole. At seven weeks the pain was so severe that a lumbar sympathectomy was performed, which immediately relieved the spontaneous pain, though the muscles

of the calf and foot remained acutely tender to pressure. At 19 weeks there were only occasional attacks of sharp stabbing pain in the foot. At 48 weeks the calf and sole remained extremely tender; motor function and cutaneous sensation were normal. He complained only of a constant mild nagging pain in the sole and toes, which was aggravated by cold and weight-bearing and lessened by heat.

CASE 4.—R.J.A. sustained multiple grenade wounds of the middle and lower thirds of the right thigh. At wound *débridement* the femoral artery and vein were ligated; the medial popliteal nerve was partially divided in the lower third of the thigh (it was not repaired), while the lateral popliteal escaped. At six weeks there was a complete paralysis of the medial popliteal motor field, with paresis of the lateral. With the exception of extension of the big toe, which remained weak, the latter field had recovered completely at the end of ten months. Function in the gastrocnemius and soleus commenced to return at ten months and eventually was good; but the long flexors of the digits displayed insignficant activity after three years. At six weeks the sole and dorsum of the foot were anæsthetic. At seven months the forepart of the sole was hyperalgesic; sensation over the dorsum of the foot had greatly improved, but was still slightly defective at the end of three years. The sole has remained insensitive to light stimuli and hyperalgesic to firm pricking.

Pain.—Two weeks after injury the patient was complaining of severe pain in the sole and deep in the foot. The pain was continuous, but fluctuated in severity and felt, he said, as though "barbed wire were being pulled through the foot". Heat and cold were without effect on the pain, which prevented sleep and required sedation. As the pain showed no signs of easing, a lumbar sympathectomy was performed at 12 weeks, resulting in immediate relief, which persisted for a year. Subsequently he had some attacks of milder burning pain, which coincided with the improvement of sensibility of the sole.

CASE 5.—J.W. sustained a perforating bullet wound of the upper third of the left thigh with complete paralysis below the knee but little soft-tissue damage or infection. At 16 weeks restoration of motor function was well advanced in all but the long extensors and flexors of the toes, which were contracting weakly; the sole was hyperalgesic, but the remainder of the cutaneous field was anæsthetic. After a year there was residual paresis of only the long extensors of the digits. During the next two years these gradually improved, but three and a half years elapsed before motor function was normal. Recovery of sensation lagged behind motor regeneration; at four years a radiating tingle was the only response to light touch and pin-prick throughout both fields.

Pain.—When he was struck the foot became numb. Immediately his boot was removed at the dressing station he was seized with a severe pain in the sole from the toes to the heel and up the inside of the leg. This pain, which was described as a "pulling pain, like a very bad toothache", persisted. There were few remissions. Heat made it worse and cold eased it; he used to put his foot outside the bedclothes to obtain relief. Ten weeks after the injury the pain was relieved for eight hours by spinal anæsthesia, but returned with its former intensity; the pain was then confined to the sole and toes. At 18 weeks a lumbar sympathectomy was performed, and for the next three months there was no pain in the foot. At 43 weeks he complained only of occasional mild attacks at night, precipitated by warmth and relieved by cold. These attacks gradually subsided, and four years after his injury there had been no return of pain.

CASE 6.—In addition to a severe head injury, C.W.J. sustained a fracture of the left pelvis in a motor-car accident. Examination three weeks later revealed paralysis of the anterior tibial group of muscles and severe paresis of the remaining muscles below the knee. The only reference to sensory function was that the patient "could feel pressure but not light touch on the toes". The anterior tibial group commenced to contract 53 to 62 days after the injury. Motor function improved rapidly; at two and three-quarter years only the long flexors of the toes remained weak. The medial and lateral popliteal fields were mildly hyperalgesic at 92 weeks and mildly hypoalgesic at two and three-quarter years.

Pain.—The patient's account of his pain dated from the recovery of his memory at five and a half weeks. The pain was felt in the foot from the ankle forwards, with maximal intensity beneath the toes. In addition, paræsthesiæ in the lower lateral popliteal field had been recorded in his history. The pain in the foot was continuous and burning; it gradually increased in intensity for a few weeks, remained at a maximum for two months, and then gradually abated. The pain would persist for hours without any reduction in intensity and for nearly three months kept him awake until 3.30 a.m. After two years the leg would swell after heavy exercise and the pain would return if the foot were raised for twenty minutes; otherwise the patient was quite free of attacks.

CASE 7.—G.W.B. sustained a perforating bullet wound of the left buttock, which resulted in a fracture of the femur and a complete lesion of the left sciatic nerve. Soft

tissue damage was severe, but infection was avoided. The medial hamstrings were contracting feebly ten weeks after the injury. Exploration at 36 weeks revealed that the nerve was in continuity, but involved in scar tissue as it crossed the ischium; it was not disturbed. Stimulation produced contractions of the medial hamstrings only. The hip joint was ankylosed. Eighty-two weeks after injury the leg was amputated because there was no prospect of recovery below the knee and the trophic changes were severe.

Pain.—Twelve days after injury the patient complained of terrific pain in the foot. The pain was continuous and of constant intensity, "like corkscrews in the foot"; it seemed to shift from heel to toes and back again. He had a sedative injection every four hours. The pain began to subside at seven weeks, but later changed to a burning sensation which was not continuous but came in attacks which occurred mostly at night. Hot water would relieve it, but he knew of no factor which would precipitate the pain. Eighty weeks after the injury he was still getting occasional mild attacks at night.

CASE 8.—H.G.T. sustained a perforating bullet wound of the left buttock with a fracture of the femur and a complete lesion of the sciatic nerve below the supply to the hamstrings. Regeneration was slow and patchy; contraction in the calf muscles appeared at 19 weeks. Eventually recovery of the calf muscles was good, but function in the other muscles was negligible. Nineteen weeks after injury both sensory fields displayed hyperalgesia of an extreme degree. At 82 weeks the sole was still acutely hypersensitive, and stimuli to the lateral popliteal field sent a shooting pain into the foot. Exploration at 134 weeks revealed that the nerve was in continuity, though surrounded by fibrous tissue. Neurolysis was performed. At 202 weeks there was no motor recovery in the lateral popliteal field.

Pain.—As soon as he was injured he felt a severe burning pain in the sole. For a time this was attributed to pressure from a plaster. Over a period of two months he had many injections of morphine. The pain continued after removal of the plaster, though it began to diminish in intensity and he no longer required morphine. Cooling the foot with methylated spirit gave a little ease. The foot always sweated a great deal (it still does after four years). The burning pain continued at diminishing intensity for two and a half years, and he thought that it disappeared finally after the exploration of the nerve.

CASE 9.—D.L.S. sustained a perforating bullet wound of the middle of the right thigh; the sciatic nerve was damaged. All the muscles were paralysed; the only reference to sensory function stated "the peroneal nerve sensory distribution not appreciative of touch". At 20 weeks sensation showed signs of recovery and the gastrocnemius was contracting. Recovery was slow but at four years a good functional result had been obtained; the medial popliteal motor field was normal and the lateral popliteal group were contracting strongly with the exception of the *extensor hallucis longus*, which was still weak. Sensation was normal.

Pain.—At the same instant as he was wounded he felt a burning sensation deep in the metatarsal region, which persisted. At 19 days he was complaining of severe "pins and needles", but the continuous severe burning pain was still present in the foot; hot salt baths afforded some relief, but morphine was administered on several occasions. Later pain became more intermittent and after six months began to ease as sensation and movements returned and improved. At three years he said that attacks were occasionally brought on by the hot north winds, and this story was repeated a year later. The affected field often perspired freely.

CASE 10.—C.G.R. sustained a perforating bullet wound of the lower third of both thighs. On the left the sciatic nerve was seen to be in continuity in a large wound, but there was a complete sciatic palsy. On the right side the wounds of entry and exit were small and a mild sciatic paresis recovered rapidly; motor function was approaching normal at 14 weeks, at which time the sole was hyperalgesic and the musculo-cutaneous field a mixture of patchy hypoalgesia and hyperalgesia. Recovery appeared in the left calf muscles, peronei and *tibialis anterior* at 13 weeks, and in the flexors and extensors of the toes at 21 weeks. Regeneration was slow, but at two and a half years there was medium functional recovery of both divisions. From the beginning of regeneration the sole had been extremely hypersensitive, while sensation in the lateral field was patchy. At two and a half years there was only slight dulling of sensibility in both fields.

Pain.—For five weeks he complained of severe burning pain in the right sole, and at six weks hyperalgesia of the foot and causalgia were recorded. Great improvement was recorded at ten weeks, though he was bothered by tingling for some time.

CASE 11.—J.R.N. sustained a perforating bullet wound through the middle of the right thigh, which resulted in complete loss of function in the sciatic field. After a few weeks pin-prick over the sole and lateral aspect of the leg produced severe pain. Exploration at eight weeks revealed an apparently undamaged nerve, which was

slightly adherent to surrounding muscles. Recovery was rapid in the muscles supplied by the medial popliteal nerve and slower in the lateral popliteal; all muscles supplied by the former were contracting 17 weeks after the injury and were normal 27 weeks later. Contraction was observed in the anterior tibial group at 31 weeks; 114 weeks later the *tibialis anterior* and peronei were contracting strongly, the remainder only weakly. During recovery the muscles were exceedingly tender to pressure. A year after the injury any mechanical stimulus to the affected skin or light pressure on a scar at the neck of the fibula resulted in severe burning pain in the sole.

Pain.—Immediately the patient was hit he experienced pain over the top of his foot, which was so intense that he thought he had been hit in the foot. This pain was likened to corkscrews being driven into the foot. It became worse three to four weeks after the injury and persisted at this peak until exploration. The pain kept him awake and required sedation. It was aggravated by movement and he liked to rest in one particular position on his side; if he could retain this position and keep still he could bear the pain. At this stage he was in New Guinea in hot weather. Other than drugs nothing was done or suggested to relieve the pain. The pain was mostly localized to the central portion of the foot, but occasionally it commenced at the metatarsal heads and then extended slowly towards the heel. During this period he stated that he had been given injections into the back to relieve the pain; these would bring relief for only a day or two. (A note in his records stated that the lumbar sympathetic had been injected with 20 cubic centimetres of "Novocain".)

Following exploration at eight weeks the entire leg was encased in plaster; the pain was about the same as before. One month later the plaster drove into the region of the fibular neck, giving a plaster sore which left a circular scar. At the same time the lower part of the limb swelled and the plaster had to be cut. With the onset of the pressure an acute burning sensation was felt in the sole. It appeared to be superficial and was very severe. It was relieved by cold and while in the hospital the patient used to leave the leg out from under the blankets and would also pour methylated spirits over the foot to cool it. This burning pain was present 145 weeks after the injury; it was practically constant, but did not keep him awake at nights. It was aggravated by a sudden fright or by attempts to regain his balance if he suddenly slipped. Cooling the part still relieved the pain. The foot perspired profusely and felt much warmer than the normal foot.

CASE 12.—T.G.W. sustained a perforating bullet wound of the left thigh with a grossly comminuted subtrochanteric fracture of the femur and complete loss of function in the sciatic field; shock and infection were severe and the wound discharged for a year, several sequestrectomies being necessary. "Almost imperceptible function of the flexors of the knee" was reported at eight weeks. The nerve was explored 47 weeks after the injury; it was freed up to the sciatic notch by dissecting away about three inches of scar tissue. There was no evidence of severance, but the disposition of strong fibrous adhesions suggested possible partial injury laterally. There was no note of nerve function at this time. Nineteen weeks later contraction of the hamstrings and calf muscles was the only sign of recovery. At three and three-quarter years the digital musculature was paralysed; the calf muscles and *tibialis posterior* were normal, while the *tibialis anterior* and peronei were contracting against resistance. At 102 weeks pin-prick over the outer aspect of the leg gave rise to a tingling sensation referred distally; 20 weeks later the entire sciatic cutaneous field was hyperalgesic with the exception of the dorsum of the foot, which was still anæsthetic. At three and three-quarter years there were no anæsthetic areas; sensation was still defective, but was better over the sole than elsewhere. Recovery occurred earlier and advanced to a greater degree in the medial popliteal field. Trophic changes were severe early, but had almost completely recovered at the later stages.

Pain.—There was no feeling in the leg for four to five days. A severe crushing, squeezing pain then commenced in the foot. It varied in severity, coming in attacks which were worse at night. The formation of abscesses in the thigh increased the pain in the foot and their drainage eased it. Morphine was administered three times daily for some time. The attacks became less severe after the exploration of the nerve and gradually subsided. They had ceased long before the sensory field of the sciatic became hyperalgesic.

CASE 13.—W.A.T. sustained a perforating bullet wound of the lower part of the left thigh, with considerable loss of muscle and surface tissue, and subsequent infection. At six weeks mild paresis of all muscles below the knee was recorded; the paresis was more marked in the medial popliteal field. At this time the lateral popliteal sensory field was hypoalgesic and that of the medial popliteal hyperalgesic. At 27 weeks motor recovery was normal, but light touch and pin-prick on the anterior part of the sole gave rise to an unpleasant tingle.

Pain.—At four weeks "pain and pins and needles in the foot" were recorded. The pain slowly increased in severity. It was described as a "dull pain" in the sole, about midway between the medial and lateral margin and extending from the heel

to the ball of the foot. It was severe at night, was eased by warmth and made worse by cold. At 16 weeks it was reported to be lessening, and at 24 weeks there were only occasional mild attacks.

CASE 14.—F.B.G. sustained a perforating bullet wound of the upper third of the right thigh, which resulted in complete loss of function in the medial popliteal field and a paresis of the lateral popliteal field, which completely recovered within a year of the injury. Recovery of the muscles supplied by the medial division was slow, but after three years was considered to be complete. At 16 weeks the skin of the sole was extremely hyperalgesic; at 11 months it was normal. There was no sensory defect in the lateral popliteal field.

Pain.—Three days after injury the patient was complaining of severe pain and pins and needles in the sole, which persisted for several months. At three and a half months the right foot would become painful when cold. At four and a half months a burning ache was continuously present in the instep. At six and a half months the pain had eased considerably.

CASE 15.—K.U. sustained soft-tissue bullet wounds on both thighs and both popliteal regions, together with a compound comminuted fracture of the right calcaneus and talus. On removal of the plaster at eight weeks there was evidence of a complete lesion of the right medial popliteal nerve which had been involved in a perforating bullet wound in the popliteal fossa. Motor recovery commenced in the gastrocnemius at 12 weeks. Eventually all movements recovered, though the long flexors of the toes remained paresed. The anæsthetic sole became extremely sensitive to pin-prick at 33 weeks and remained so for three years. The intrinsic muscles of the foot wasted profoundly, and trophic cutaneous changes were observed.

Pain.—Burning sensations commenced in the foot three to four weeks after the injury; he thought the plaster might have been responsible. The pain started below the medial malleolus and spread forward through the foot to involve all the toes. Sometimes it came in frequent brief attacks which would wake him at night, and sometimes it was continuous, severe and burning. Attacks were precipitated by hot days. Removal of the plaster at eight weeks gave relief which lasted only a day, when the pain returned. Within a few weeks the attacks became mild and infrequent and finally disappeared.

CASE 16.—D.M. received an extensive soft-tissue perforating shrapnel wound of the right popliteal fossa, which was excised, and the limb was enclosed in plaster. A complete lesion of both sciatic divisions was noticed on removal of the plaster two weeks later. Profound wasting occurred, with trophic changes in the foot. At 12 weeks cutaneous sensation was patchy, especially over the sole, where hyperalgesia and hypoalgesia alternated in an irregular fashion. At 29 weeks the entire field was acutely hyperalgesic; hypoalgesia replaced hyperalgesia in the lateral and medial popliteal fields at 38 and 87 weeks respectively. Cutaneous sensation was almost normal at four and a half years. At 15 weeks recovery commenced in the motor field of the medial popliteal, and at 19 weeks in the lateral popliteal. Motor function (excluding the intrinsic musculature) was normal at four and a half years.

Pain.—When he was hit he felt a severe pain in the knee. On his attempting to stand this pain extended into the leg and foot; it was severe and was described as "gnawing" in character. About three hours later a burning quality was added and this was felt along the outer aspect of the lower leg and foot. After wound excision some hours later the burning pain settled in the foot, where it was maximal about the ankle and toes. The pain was continuous and severe and required morphine sedation. Hot sea baths on the hospital ship relieved the pain; at the base hospital heat therapy was found to relieve and cold to aggravate the pain. The foot perspired freely in severe attacks. The pain along the outer aspect of the lower leg persisted for about 11 weeks, when it was said to have been "cured" by massage. The burning pain in the foot persisted, but commenced to subside spontaneously a few weeks later; at eight months it had ceased to trouble him except in cold weather.

CASE 17.—A.H. sustained a perforating bullet wound of the left popliteal region which resulted in paralysis of, and patchy loss of sensation over, the lateral popliteal field. Function was mildly impaired in the medial popliteal field, but rapidly recovered. Motor recovery appeared in the peronei and *tibialis anterior* at 18 weeks; the extensors of the toes were contracting 12 weeks later. Motor recovery at 15 months was good, but over the cutaneous area there was an unpleasant hyperalgesic response to mechanical stimuli. At two and a half years motor function was normal but for a mild residual paresis of the toe extensors, while sensation was still mildly defective over the dorsum of the big toe and adjacent portion of the foot.

Pain.—The day after he was wounded he felt a burning, stinging pain in the big toe. The pain was constant and severe and remained for many months. At 15 months he complained only of attacks in very cold weather. Two and a quarter years after the injury the lateral popliteal nerve was tender to palpation at the neck of the fibula, but there was no spontaneous pain.

Plexus Lesions.

CASE 18.—J.I.C. sustained a perforating bullet wound at the right axillary outlet with injury of the brachial plexus and division of the axillary artery. All the muscles of the hand and forearm were paralysed, while the biceps, brachialis and brachioradialis were paresed. The forearm and lower third of the arm were anæsthetic. Sensation improved rapidly. At five weeks the anæsthesia was confined to the ulnar field and six weeks later had been further reduced to a narrow zone along the ulnar margin of the hand and little finger. Sixteen weeks after the injury the palmar aspect of the hand and digits was acutely hyperalgesic; this hyperalgesia slowly subsided, but was still evident three years after the injury. One year later the median field was still hyperalgesic and the ulnar field was mildly hypoalgesic. At 11 weeks all muscles, with the exception of the ulnar half of the *flexor digitorum profundus* and the ulnar intrinsic muscles, were contracting—the proximal musculature against gravity and the distal only feebly. Power improved rapidly, but there was no contraction in the *flexor digitorum profundus* and ulnar intrinsic muscles for a year. When last examined four years after the injury motor function was very good.

Pain.—Twenty-four hours after the injury a constant burning sensation was felt in the hand and fingers. The entire forearm became tender and the patient could not bear to be touched; sedatives were used freely for the intense pain. The pain was maximal in the hand, was constantly present, but fluctuated in intensity. At ten weeks there was some improvement, and he complained of pain in the fingers only; the hand was purplish red in colour, with hyperkeratoses over the palm, and it perspired freely. The pain then rapidly subsided and disappeared, but the palm and the palmar surfaces of the fingers remained acutely hyperalgesic for three years.

CASE 19.—D.T.A. sustained a perforating bullet wound of the left axilla. The wound of entry was just below the clavicle and the surgical neck of the humerus was fractured. The lesion of the plexus was complete, but later presented the features of damage to the medial and lateral cords. Recovery commenced within three weeks in the circumflex and radial nerves, and in four weeks in the forearm muscles supplied by the median and ulnar. At four years recovery was poor in the intrinsic muscles of the hand, but good elsewhere. The predominant sensory loss in the hand was in the median field, with a strip three to four inches wide running up the anterior surface of the forearm and arm. After four years some diminution of sensation could still be detected in the whole of this area. The involved field was never hyperalgesic. Within the first few months trophic changes were profound, the skin of the hand being shiny and pink. These soon cleared.

Pain.—Immediately the patient was injured he felt pain throughout the arm, which was most severe in the forearm and palm. He could get no sleep, but rubbing the hand gave some relief. Three days later he felt in addition a sensation of "pins and needles" in the back and front of the hand and fingers, with a throbbing quality. The most severe attacks of pain occurred at night and he needed morphia to ease it. The pain was very severe for two months, then gradually lessened for two to three months and finally disappeared.

CASE 20.—W.H.E. sustained head injuries and damage to the left plexus when he was thrown out of a truck. There was complete paralysis of the arm, while the skin below the elbow and over the dorsal surface of the upper arm was anæsthetic. Horner's syndrome was observed for three years and the hand never sweated. The deltoid and the flexors and extensors of the forearm showed some recovery, but two and three-quarter years after injury the arm was useless and amputation was performed.

Pain.—On recovery of consciousness at two to three weeks the patient complained of constant "crushing" pain along the ulnar border of the hand and the ulnar two fingers, which caused much loss of sleep. The pain gradually lessened, but did not disappear. At two and three-quarter years he was having daily a "grinding" pain in the ulnar border of the hand, which was worsened by activity and eased when he rested at night. During the day the pain could be eased by flexion of the neck. Three months after amputation he claimed that the operation had given great relief, though he still felt a mild pain in the two ulnar fingers of a phantom hand. The relief suggested that the weight of the paralysed limb was a responsible factor in causing pain.

CASE 21.—A.D.R. sustained a large gunshot wound of the left axilla in which two foreign bodies were retained. An operation was reported to have been performed "to remove obstruction to the ulnar nerve". No details were provided. The medial cord of the plexus was damaged and resulted in a complete ulnar palsy and partial median paralysis (the *pronator teres* and *flexor carpi radialis* were functioning). Motor recovery in the median field commenced at eight weeks and was complete at 38 weeks. Contraction was observed in *flexor carpi ulnaris* at 38 weeks, in the hypothenar muscles at 81 weeks and in the first dorsal interosseous at 85 weeks. At eight weeks the

cutaneous fields of both nerves were hyperalgesic; at 40 weeks the median had recovered, but the ulnar field was still hypersensitive. At 117 weeks sensation was almost normal and motor recovery was satisfactory. Trophic changes were profound early but improved later.

Pain.—The day after the injury the patient began to suffer from sharp attacks of agonizing pain in the elbow, in one of which he fainted. These attacks were relieved only by morphine. The hand was numb at first and the pain was not felt below the elbow, but after a week a continuous burning, stinging pain was felt along the ulnar border of the forearm and all over the hand. At 23 weeks it was localized to the ulnar field and occasionally along the inner border of the forearm. It was relieved by cold and worsened by heat and by excitement. After one and a quarter years the pain began to subside, but at two years "red-hot" bursts in the hypothenar region could be precipitated by emotional stimuli (for example, looking down from a height).

CASE 22.—W.A.M. received large shrapnel wounds of the left axilla and inner aspect of the arm, which required skin grafts. The wounds were grossly infected. The motor fields of the radial, median and ulnar nerves were severely paresed and there were mixed zones of hyperalgesia and hypoalgesia below mid-humeral level; the palm and digits were acutely hypersensitive. At 121 weeks sensation was almost normal. At the last examination, 280 weeks after the injury, there had been some further sensory improvement, while the residual motor defect was negligible. Trophic changes were originally severe, but recovered. Palpation of a neuroma above the medial epicondyle indicated that there was an additional lesion of the ulnar trunk.

Pain.—Three days after injury a constant deep burning pain commenced. This would radiate from the ulnar side of the wrist into the palm and into the bases of all the fingers and to the tips of the fourth and fifth digits. Exacerbations of pain were caused if the hand was knocked, by breathing deeply, by heat, exertion or loud noises, and tickling his feet would "nearly cause the hand to drop off". The pain was much worse during the day than at night. This he attributed to the glaring sunlight in the desert. The hand was flushed and perspired freely. Cooling relieved the pain. He used to carry a wet rag in his palm for this purpose. At 40 weeks the constant pain was replaced by a painful tingle and the exacerbations became less frequent; severe attacks of lancinating pain would, however, occur in hot weather. At 280 weeks slight pain was still felt if he became hot and sweated, if the hand became cold, or upon taking a deep breath.

Median Nerve.

CASE 23.—R.H.L. sustained a penetrating bullet wound of the left arm 12·5 centimetres above the medial epicondyle and, the brachial artery having been damaged, a tourniquet was applied. There resulted a generalized muscular paresis below the elbow which was maximal in the median field. Defective sensation was observed over the whole extent of the forearm and hand. The latter showed trophic changes, was pink and perspired freely. Recovery was well advanced at 14 weeks, there being only a mild paresis of the muscles supplied by the median nerve. Loss of sensation was limited to the dorsum of the hand and a strip running up the dorsum of the forearm. The whole palmar surface of the fingers and hand was excessively sensitive—after a shave he could feel the bases of the hairs as sharp points with the left hand when the right could not feel them.

Pain.—From the beginning a constant severe burning pain was felt in the thumb and index finger, which could be eased temporarily by warmth. At 27 weeks the pain was sufficiently severe to call for surgical treatment. The median nerve and brachial artery were exposed. The nerve appeared normal. An attempt was made to strip the artery, which was very small and involved in scar tissue. The vessel pulsated only as far distally as the scar, in which dissection revealed a large aneurysmal sac on the posterior aspect of the artery. The involved segment and a neighbouring foreign body were removed. This operation failed to relieve the pain. Three weeks later preganglionic sympathectomy gave immediate, complete and permanent relief, but did not diminish the hyperalgesia.

CASE 24.—N.B.P. sustained a partial median nerve lesion from a tangential bullet wound at the level of the right axillary outlet. Within 12 weeks motor function was approaching complete restoration. Initial complete median anæsthesia was replaced in four weeks by hyperalgesia. At 54 weeks there was some general dulness of sensation in the median area; all movements were full in range and could be correctly executed against strong resistance.

Pain.—Fifteen minutes after injury he felt excruciating pain beneath the nails of the thumb, index and middle fingers, which caused him to faint. After 11 hours the pain subsided, but returned in a few hours. It was felt both deeply and superficially throughout the fingers; its continuous burning was worsened by exercise, by noise of planes, or by shelling. Sometimes the pain was relieved by warm water. He could

not sleep. The hand was reddish in colour and was warm to the touch. Preganglionic sympathectomy performed six weeks later was followed by complete relief. At four months, however, he complained of minor transient attacks of pain shooting from the elbow along the palmar surface of the forearm; the hyperalgesia had not lessened. At 54 weeks the only residual sensory defect was slight hypoalgesia of the terminal phalanx of the thumb, index and middle fingers. Relief from pain was complete and permanent.

CASE 25.—N.N.P. sustained a tangential bullet wound of the lower third of the inner aspect of the right upper arm, which exposed the brachial vessels and caused a complete paralysis of the median nerve. Recovery appeared in the forearm muscles 29 to 33 weeks after injury and in the thenar muscles at 38 weeks. Motor regeneration progressed slowly. At 83 weeks all muscles were contracting to give a full range of movement against resistance; the index finger was anæsthetic and the palm hyperalgesic. The nerve was explored at 90 weeks, freed from scar tissue, and "a large lateral bulb removed".

Pain.—Two days after the injury he complained of severe pain running from the wound to all the fingers, and at six days "mild causalgia" was recorded. This soon subsided. At seven weeks, when a plaster case was removed, the attacks recurred. Immediately the forearm and hand warmed up (for example, as under a lamp at physiotherapy) a burning pain would radiate from the wound down to the thumb and the radial side of the palm. The pain was maximal in the thumb and thenar region. During attacks the hand would go red and perspire freely. He could relieve the attacks by cooling the hand. After discharge from the army he had to give up his position as a pastrycook because of the deleterious effects of heat.

CASE 26.—A.J.G. sustained a perforating bullet wound of the lower third of the left upper arm, which resulted in a comminuted fracture of the shaft of the humerus at the junction of the middle and lower thirds. The patient immediately noticed numbness in the forearm and hand. There was severe paresis of the muscles supplied by the median nerve at 13 weeks; improvement was noted at 16 weeks and good recovery occurred within 24 weeks. At 13 weeks the median cutaneous field was hyperalgesic with anæsthesia in that of the medial cutaneous nerve of the forearm; at 24 weeks the median field had almost recovered, but the latter had not improved. At 13 weeks the hand was slightly swollen with pinkish-blue mottling and patchy desquamation. These trophic changes rapidly cleared.

Pain.—When the numbness subsided a continuous pain developed in the cutaneous distribution of the median nerve. At 18 days "severe causalgia in index finger" was recorded. At 13 weeks the pain began to ease and for a time was felt only in the thumb and the tip of the index finger. The pain was made worse by cold, exercise or mental shocks; he likened it to toothache. At 16 weeks there were only occasional mild attacks, and eight weeks later these had ceased.

CASE 27.—H.O.E. sustained a perforating bullet wound of the lower third of the left upper arm with an incomplete motor lesion of the median nerve. Sensation was defective in the palmar field, while the radial three and a half digits were anæsthetic. Within 30 weeks motor function was approaching normal. The palmar field was hyperalgesic at 11 weeks. At 14 months sensation was absent in the digits, but was almost normal over the palm. At four years sensation was only slightly defective over the digits. Trophic changes caused spindling of the fingers. At 11 weeks a neuroma was palpable at the site of injury.

Pain.—Fourteen days after the injury a constant "nagging" pain was felt deeply and superficially in the hand. It kept the patient from sleeping. The pain was severe for a month and then started to improve, coming in one or two bouts per day, each lasting about two hours. Cold and exercise (as at occupational therapy) precipitated attacks and a slight blow on the wound in the arm would precipitate pain in the hand. The hand perspired so freely during the attacks and during exercise that he had to wipe it. The pain ceased at ten months, but he had one attack, lasting approximately 30 minutes, about two and a quarter years after the injury.

CASE 28.—L.C.P. sustained a clean perforating bullet wound of the left arm 20 centimetres above the elbow, and a more severe bullet wound of the right cubital fossa. Median nerve function was only mildly impaired on the left and recovery was complete in six months. There was a complete lesion of the right median and the right brachial artery was occluded; there were severe tissue loss and infection with gas gangrene. The patient was seriously ill and in pain for a long time. Most of the muscles supplied by the right median were contracting at 32 weeks, but subsequent recovery was slow. The nerve was explored at 51 weeks and freed from dense scar tissue. Recovery progressed slowly and at three years there was moderate function in all the muscles. At six months the median field was still anæsthetic. The area gradually became hyperalgesic. At three years pin-prick was correctly localized, but gave a burning sensation; light touch was perceived. At 283 weeks sensation was still

defective over the whole of the median area, with touch absent over the index and medius. Trophic changes on the right were severe, with spindling of the fingers, while there were no such changes on the left (see Figure I).

Pain.—Severe pain in the left hand commenced two weeks after the injury, but there was never any such pain on the right side, which was badly injured. The patient said that he suffered "hell" for four months and at 32 weeks the pain was recorded as "severe". The pain used to subside to half-intensity in the darkness and when the sun rose increased again to continuous pain of a severe aching character, which would make him lie still and cry. He used to keep a damp cloth in the palm of the hand, but this gave little relief. He was sure that light rather than heat was responsible for the intensification, because it occurred soon after sunrise on bright, cool mornings

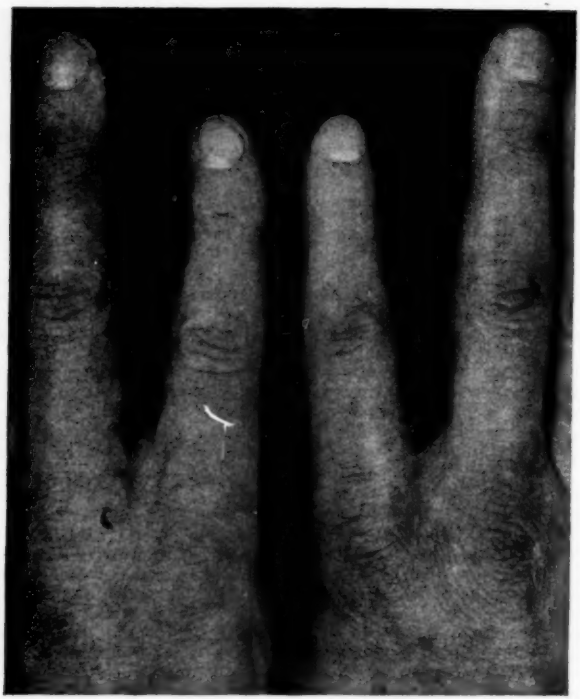

FIGURE I. Case 28. Bilateral median nerve lesion 283 weeks after injury. The left index finger (mild lesion, causalgia) is normal, while there are severe trophic changes on the right (severe lesion, no causalgia).

in the desert. Visual shocks brought on exacerbations (for example, seeing a man on crutches trip over, or seeing two motor-cars nearly collide). The pain gradually eased, but for 18 months he was susceptible to emotional stimuli, and at 116 weeks occasional attacks of severe causalgic pain in hand and fingers were recorded. At 283 weeks fatigue or worry would bring on an ache in the index and medius which would extend up the forearm. The left hand still sweats more than the right.

CASE 29.—D.J.P. sustained a perforating shrapnel wound just above the right elbow and suffered severe loss of tissue, with infection, which necessitated skin grafts later. The brachial artery was divided and the median nerve completely paralysed. At five months recovery had commenced in the forearm muscles. At ten months the *flexor pollicis longus* and the section of *flexor digitorum profundus* which supplied the index were still paralysed, while the other muscles were still paresed. Final recovery at four and a half years was fair, except in the long flexors of the thumb and index finger. At five months the palm was hypoalgesic and the radial three digits were anæsthetic. Three months later the palm became hyperalgesic. At two

years there were small areas of anæsthesia at the tips of the thumb and index finger; elsewhere sensation was slightly reduced with patches of hyperalgesia. At four and a half years hyperalgesia in the palm remained acute. In addition there were large areas of skin on the forearm and arm, stimulation of which caused pain to radiate to the palm. Trophic changes were severe from the beginning and had improved little at four and a half years. The digits were spindled, shiny, cyanotic and cold. At one of his operations the plastic surgeon observed the nerve to be in continuity, though with a neuroma.

Pain.—The hand went into a cramp when he was hit and then went "dead". A tourniquet was applied and left on for about five hours. Two days later the patient had a sudden attack of severe pain throughout the hand, which seemed to be on fire. This lasted for five minutes. Agonizing attacks then recurred several times a day. He was given injections to control the pain. In one attack the hand seemed to be above his head when it really was powerless by his side. The burning pain was worst over the ball of the thumb, but in severe attacks would spread to chest, body and eyes. The attacks became longer in duration and would last up to five hours. They most frequently occurred in the afternoon and at night. At first he thought the pain was due to the plaster in which his arm was encased, for it improved at every change of plaster. After the last plaster was removed the attacks continued, but were not as severe and were less frequent. Pain could be precipitated by noise, especially when caused by aircraft. The hand went red in attacks and would sweat profusely. Cold water had little effect on the pain, but hot water would relieve it for about ten minutes. The hand was very sore after exercise. In attacks he would rest the hand motionless on a pillow, guarding it against the slightest stimulus. He was still having occasional attacks at 45 weeks, and at 65 weeks the hand would smart and burn in hot weather. At 102 weeks an attack was brought on by seeing a man narrowly avoid falling from a tram. At four and a half years he said that he occasionally felt pain in the hand; it had a pricking rather than a burning quality. It could be brought on by emotional or visual stimuli or by a scraping sound.

CASE 30.—C.G.M. sustained a perforating bullet wound of the left upper arm, which resulted in complete loss of function in the median and ulnar fields. The wound became infected. The *pronator teres* and *flexor carpi radialis* were contracting five days later. The onset of recovery was delayed in the remaining muscles, but four years after the injury all muscles were contracting against strong resistance. Sensory recovery was observed in the palm at five days, but six weeks elapsed before the entire field was sensitive. Hyperalgesia was never recorded. At six months sensation was only mildly impaired in the median field. Trophic changes were a characteristic feature. They appeared early and, despite the good sensory recovery, were still pronounced four years after the injury. The palm and radial three digits were red, the nails long and deformed and the digital pads wasted.

Pain.—The patient was wounded at dusk and severe burning pain commenced immediately in the radial half of the hand. It continued for three months, was always confined to the median field in the hand, fluctuated in intensity, but was definitely worse when the arm was septic. It gradually subsided on the hospital ship while the patient was being transported from the Middle East to Australia. When the pain was severe, relief could be obtained only by sedation.

CASE 31.—A.K. sustained a clean perforating bullet wound of the right arm; the brachial artery was severed and conduction interrupted in the median and ulnar nerves. Signs of recovery were evident in the median field within 15 weeks. Motor function was fully restored 49 to 86 weeks after the injury. At the latter date the median cutaneous field was extremely hypersensitive. When last examined at 114 weeks sensation was normal. Trophic changes were mild in the initial stages, but had cleared at six months.

Pain.—Pain commenced in the palm within one hour of the wounding. It was constant, fluctuated in intensity and was described as "an agonizing toothache pain". It was very severe for a week and was always centred in the palm; it kept him awake and required morphine sedation. It then commenced to ease, with severe attacks coming only at night. Placing the hand in warm water gave temporary relief. Three months after the injury he was getting only occasional attacks—he did not know of any precipitating factor. When last examined he was still getting an attack about every two weeks, though these were not severe enough to disturb him.

Radial Nerve.

CASE 32.—W.G.R. fell ten feet and fractured the right humerus at its middle. Complete radial paralysis was reported ten days later. Motor recovery appeared in 16 weeks and progressed to completion over the following year. The web between the thumb and index finger was anæsthetic and a partial sensory defect extended over the

dorsum of the thumb and hand, but not to any fingers. Sensation commenced to recover 20 weeks after the injury. The involved area gradually shrank; at 109 weeks there was a small area of defective sensation, measuring 9·0 square centimetres on the web between thumb and index finger. He said that this area would go white and very numb if put into cold water.

Pain.—From the time of injury a burning sensation was felt in the field of the superficial radial nerve. The area felt as though it were being scalded with hot water, and so vivid was the feeling that he used to touch the part to see if it was hot, but it was not. After five weeks an improvement commenced. Later the pain was felt only when the part was warmed in bed. At 20 weeks the attacks of burning pain had ceased and there was only an occasional feeling like ice-cold water on the area.

CASE 33.—B.E.M. was involved in an automobile accident in which he sustained a comminuted fracture of the upper third of the shaft of the right humerus with complete paralysis of the radial nerve. The nerve was explored at 24 weeks. It was found in continuity, though bound in fibrous tissue and with two neuromata one inch apart. The nerve was freed. There was no recovery 13 weeks after the operation. All muscles were contracting 27 weeks later, and eventually achieved recovery of moderate degree. The extent and depth of the cutaneous sensory disturbance were slightly reduced in the initial stages and then remained unchanged until the last examination three and three-quarter years after the injury. At 14 weeks the hand was cold and cyanotic, with œdema of the dorsum. These changes soon disappeared.

Pain.—Immediately after the accident he felt a continuous burning sensation over the back of the hand and wrist. This continued for two months, when it began to come in intermittent attacks. Eventually it subsided. At 14 weeks deep pressure on the radial nerve at the elbow gave rise to a burning sensation in its cutaneous field.

Ulnar Nerve.

CASE 34.—R.R.V. had his left brachial artery and ulnar nerve severed in a grenade wound which caused extensive laceration of the mid-humeral region. A tourniquet was applied and the artery was ligated. The patient was very ill for a long time, with severe infection and much pain. The nerve was sutured and transposed anteriorly 314 days after the injury. The *flexor carpi ulnaris* and *flexor digitorum profundus* commenced to contract 11 to 26 weeks after repair; 15 weeks after the latter date the hypothenar group was contracting. Two years after the operation all the muscles were contracting against resistance—the range of activity was restricted by extraneural changes. At the beginning there was complete sensory loss in the ulnar and medial cutaneous of the forearm fields. At 22 months there was no change in the hand, though improvement had occurred in the forearm. At two years (61 weeks after suture) there was patchy restoration of pain sensation in the hypothenar area with a tingling radiation. No recovery occurred in the little finger.

Pain.—Three days after the injury the forearm and then the hand became painful; at this time the records stated that there was "no sensation in hand or forearm". For three months the patient suffered from very severe pain, which was described as "neuralgic", in the whole of the hand and the lower half of the forearm. He had frequent injections of morphine for many weeks. The pain had disappeared before the suture was undertaken.

IV. THE CLINICAL FEATURES OF THE PAIN.

1. *Quality.*

In Denmark's[1] case the pain in the hand and fingers had a burning quality which caused great distress. Weir Mitchell[2] thought this to be the outstanding symptom in his cases. His patients usually were at pains to keep the part cool and some of them kept it carefully wrapped in a damp cloth. A similar manœuvre was adopted by the patients in Cases 22 and 28 of this series.

Burning is the description most frequently applied to the pain,[16][22][23][24][25] though it may be described in other terms.[16][25] In 21 of the present 34 cases the pain was recorded as "burning". In some it was described as a "stinging" or "tingling pain". These descriptions are believed to apply to superficial rather than to deep pain. In many cases deep paroxysms were added to the continuous sensation of heat or the pain was felt continuously both deeply and superficially. In those cases in which there is no mention of cutaneous pain (burning, tingling, stinging *et cetera*) the deep component was variously

described as "stabbing", "crushing", "tearing" or as a "severe ache". The sufferings of the patient were often very severe, the paroxysms being described as "agonizing" or "excruciating". In the initial stages the pain was usually constant, rarely intermittent, and was subject to exacerbations. At a later date, when the causalgia was subsiding the pain tended to come in intermittent attacks.

Though many patients were emphatic that cold eased the pain, several claimed that warmth gave relief and cold made it worse. A history of the effects of temperature changes was elicited in 24 cases.

Relieved by warmth, aggravated by cold	4 cases.
Relieved by cold, aggravated by warmth	5 cases.
Relieved by cold	2 cases.
Relieved by warmth	5 cases.
Aggravated by cold	4 cases.
Aggravated by warmth	2 cases.
Hot baths relieved the pain in the early stages, but at a later date hot winds aggravated it ..	1 case.
Temperature changes without effect	1 case.

Other observers have also described a variable response to temperature changes, though the majority of patients benefited from cool, moist conditions.[15][22][24][25] Ross[26] found all of his patients to be sensitive to medium degrees either of cold or of heat. A low temperature, he said, gave an aching sensation, while warmth caused a burning pain.

Sharpey-Schafer,[5] who subjected himself to section of cutaneous nerves, each time felt burning pain in the denervated area. Lewis[8] identified the skin as the site of burning pain. He was able to show that cutaneous pain had only two qualities: (*a*) "pricking" if of brief duration, (*b*) burning if prolonged. Pain from the deeper tissues he held to be entirely unlike these and to be more fitly described as "aching" or "neuralgic" in character. Earlier, Lewis, Pickering and Rothschild[27] had reported experiments which suggested that the spontaneous sensation of tingling arises in tactile fibres.

It has been suggested that the sensation of heat may be due to the increase in the circulation which frequently accompanies it.[15] In Case 32 the burning quality had no relation to the temperature of the skin; the patient was often surprised when, on touching the affected area with his finger, the skin was not hot. In 12 cases of this series the affected limb would flush and sweat more than the other (see Cases 8, 11, 16, 18, 19, 22, 23, 24, 25, 27, 28, 29), but in many others the parts were colder. Speigel and Milowsky[22] reported that their patients had "red, cold, profusely perspiring skin". The observations of Ulmer and Mayfield[30] indicate that alteration of blood flow is not responsible for the pain. The weight of evidence suggests that the abnormal sensation of heat is fundamentally due to a disorder of sensory mechanisms rather than of vascular control.

2. *Distribution.*

In every case the pain was felt in the hand or the foot and involved the distribution of the injured nerve. The continuous burning pain was usually felt superficially in the skin of the palm, sole and digits, while the "crushing" and "tearing" pains were felt more deeply. When severe the pain always radiated and came to occupy a larger area. There was no instance of the radiation of pain to other limbs, as reported by Mitchell[2] and Sauer,[28] but in Case 29 the pain spread to the patient's chest and eyes. When causalgia accompanied complete interruption of conduction, it was referred to the denervated area.

3. *Hyperalgesia.*

Areas of returning sensation are often hyperalgesic for varying periods. In the causalgic cases the hyperalgesia was usually of greater degree and more persistent, and sometimes extended over an area much greater than the field of the injured nerve.

4. *Time of Onset.*

Many patients insisted that the pain in the extremity commenced at the moment of receipt of the wound. Thus a soldier will say that he thought he was hit in the foot or the hand. On more careful questioning, however, the impression will often be gathered that the patient has not a clear memory of events at the time of wounding. He may admit that, though he was not certain the pain came on immediately, it certainly commenced within an hour or two. Three patients (Cases 5, 12, 26) said that the whole limb went numb at the moment of wounding and pain was felt as the numbness wore off in a few hours or a few days. Others were very ill for a time with infection or having bother with a severe comminuted fracture. In Cases 6 and 20 the patients were unconscious for two to three weeks and complained of pain on recovery of consciousness. In the records of several cases pain is mentioned a week or more after the injury, but the exact time of onset is not stated. The approximate times of onset in the 34 cases were:

Under six hours	14 cases
Six to twenty-four hours ..	3 cases.
One to seven days	8 cases.
Four to twenty-eight days ..	9 cases.

According to Weir Mitchell,[2] "pain may be continuous from the outset, but more commonly the neuralgias date from the time of the traumatic fevers or even still later". Recent writers,[16 to 25] however, all report an immediate or very early (within a few hours) onset in the majority of cases.

5. *Precipitating Factors.*

Precipitating factors can be graded into two classes: (*a*) the factors which determine the onset of pain; (*b*) the factors which bring on exacerbations of pain.

The Factors which Determine the Onset of Pain.—In addition to the injury to one of the more susceptible nerves, some other factor is necessary, for the great majority of such injuries are not followed by intractable pain. The bare recognition of the nerve injury as an essential causative agent does not bring us any nearer to the discovery of the mechanism by which the pain is generated. The question of the nature of the mechanism must be left for later discussion.

Factors Responsible for Exacerbations of Pain.—These can be divided into two classes: (*a*) anything which increases the activity of the limb; (*b*) anything which increases the activity of the central nervous system. It is clear that a vast number of stimuli can be included in these categories and indeed in a severe case the patient will experience a painful response to a great many agents. Any form of stimulus to the limb, such as mechanical stimuli, heat, cold, muscular activity and movement, may increase the pain. If hyperalgesia is severe even a light touch may cause intense agony (for example, Cases 2, 18 and 22).

The central nervous factors include auditory, visual and psychic stimuli, fatigue, mechanical stimuli to other parts of the body and so on. Most of the visual and auditory stimuli mentioned in the case records probably acted as emotional stimuli: looking down from a height (Case 21), loud noises (Case 22), noise of planes (Cases 24 and 29), shelling (Case 24), witnessing

a mishap in the street (Case 28). In both Cases 22 and 28, however, the patients are certain that their pain was more severe in sunlight, being particularly severe in the bright cool early morning of the desert. Thus the light rather than the heat was responsible. In two-thirds of the cases reported by Kirklin *et alii*,[25] the patients were "relieved by nightfall". In Case 29 a scraping sound could bring on the pain after four years. In Case 22 the patient at one stage felt severe pain in the hand if his foot was tickled.

In seven cases such a response to central stimuli is recorded; in six of these (Cases 21, 22, 24, 26, 28, 29) the pain was in the hand and in only one (Case 11) in the foot. Kirklin *et alii*[25] have also observed that causalgia in the lower extremity is less affected by such stimuli than in the upper extremity. A possible explanation is that the sensory centres devoted to the hand are more extensive than those related to the foot.

6. *Duration.*

The duration of the pain varied widely from case to case and, in many, was not easy to assess because of meagreness of data and the fluctuation of symptoms. In most cases the pain slowly waned, paroxysms becoming less and less frequent and their intensity less severe. It was usually difficult to say at what point the pain could no longer be called causalgia. Many patients, who had been relieved for more than a year, declared that occasional bouts of pain could be brought on by such factors as excessive cold, oppressive heat, unusual exertion or an emotional upset.

In more than half of the cases the pain had subsided to become bearable within four to five months, and in half of the remainder it did not last more than a year. Often it lost its burning quality and assumed the character of a deeper ache brought on by cold or by muscular activity. After more than five years the patient in Case 1 continues to complain of attacks of pain; and in Case 20 (complete plexus lesion) the patient did so until his amputation at three years. Both patients, however, declared that they could sleep at night.

In the 34 patients the approximate duration of symptoms was:

5 to 13 weeks	..	15 cases.
13 to 26 weeks	..	8 cases.
26 to 52 weeks	..	8 cases.
1 to 2 years	..	3 cases.

V. COINCIDENT FACTORS AND THEIR RELATION TO PAIN.

1. *Identity of Nerve Injured.*

In the vast majority of cases causalgia followed lesions of the sciatic nerve, the brachial plexus and the median nerve (see Table 1). A similar incidence has been reported by most of the previous writers on the subject. These three tracts, therefore, must possess features which render them peculiarly liable to causalgia. The duty of the investigator is to search for these features.

(i) *Sciatic Nerve.*—Pain followed injuries of the sciatic nerve or its popliteal divisions in 17 of the 34 patients with causalgia. This proportion is higher than that of some recent writers.[16][22][23][24][25] The lesion was above the level of the head of the fibula in all 17 cases. It is the experience of most observers that the lateral popliteal division usually suffers greater damage than the medial. This was so in the present total series of sciatic lesions, which have been tabulated as regards medial or lateral popliteal preponderance:

Effects greater in the medial popliteal field	..	..	..	12
Effects greater in the lateral popliteal field	..	..	..	39
Effects approximately the same in both fields		..	..	16

In nine cases the pain was felt in the sole, and though in the remainder it was diffusely spread through the foot, the history and sequence of events left little doubt that the medial popliteal lesion was responsible. The evidence indicates a much greater tendency for causalgia to follow median than lateral popliteal damage.

Level of Lesion and Precipitating Factor.—In all 17 cases causalgia was due to lesions above the head of the fibula; 16 of the 17 were due to gunshot wounds and, of these, 14 were due to perforating bullets. Table II indicates

TABLE II.

Levels of Lesions (as far as Head of the Fibula) of the Sciatic Nerve and its Branches.

Level.	Non-Painful.	Causalgia.	Total.
B	8	3	11
U/3	5	6	11
M/3	10	2	12
L/3	27	6	33
Total	50	17	67

B = buttock; U/3, M/3 and L/3 = upper, middle and lower (as far as head of fibula) third of thigh respectively.

that 22 lesions in the buttock and upper third of the thigh presented nine cases of causalgia, while the remaining 45 lesions, situated further distally, presented eight cases of pain. The apparent preference of causalgia for high lesions, therefore, is not to be explained by the greater frequency of such lesions.

(ii) *Brachial Plexus.*—Causalgia followed plexus lesions in 5 out of 25 cases (4 out of 15 gunshot wounds). The gunshot cases presented pictures with similar general features, in which the medial and lateral cords seemed to have sustained the most damage; later they assumed the appearance of combined partial lesions of the median and ulnar nerves. In Case 20 (a severe stretch lesion) irreparable damage to the entire plexus resulted.

TABLE III.

Type.	Number of Cases.	Causalgia.
Upper	13	0
Lower (including total plexus) ..	12	5

Injury to the upper roots or upper trunk was as frequent as injury to the lower trunk or medial cord. Yet causalgia did not occur in any case in which the lower fibres escaped. Dividing the series roughly into "upper" and "lower" groups, the occurrence of pain was as shown in Table III. Thus the incidence of causalgia was very high indeed in injuries of the lower part of the plexus, and in the majority the main damage had fallen on the ulnar fibres. Most writers have reported a similar ulnar preponderance in plexus causalgia. This may be related to the fact that the lower trunk and medial cord contain all of the ulnar fibres, but only a proportion of those destined for the median; these median fibres, however, are destined for the hand.

The predominance of ulnar damage was accompanied by a parallel distribution of pain. In three out of the five cases the pain was felt predominantly in the ulnar side of the hand. The frequency of an ulnar

distribution in plexus lesions contrasts strongly with its rarity in lesions of the ulnar nerve in the arm and the forearm.

In Case 20 a complete plexus paralysis resulted from a severe stretch lesion, and Horner's syndrome indicated that the sympathetic had been injured. Paget[29] described this syndrome in two stretch lesions, but André-Thomas[30] was the first to attribute significance to the combination of sympathetic injury with causalgia. The earliest recorded case of plexus causalgia following stretch is that of Earle[31] in 1816.

(iii) *Median, Ulnar and Radial Nerves.*—The median nerve claimed the great majority of the cases in which pain followed injuries to the upper limb (Table IV).

TABLE IV.

Nerve.	Number of Cases.	Causalgia.
Median	67	9
Ulnar	91	1
Radial	63	2

As in the leg, causalgia manifests a decided preference for nerve injuries in the proximal segment. In this series there were no cases of causalgia in injuries below the elbow.

(iv) *Further Selection of Cases for Subsequent Discussion.*—Most of the future argument will relate to lower plexus lesions, median lesions above the elbow and sciatic lesions above the head of the fibula, which, though comprising only 114 of 312 nerve lesions (36%), supplied 31 out of 34 cases of causalgia (91%). By the exclusion of ulnar and radial nerve lesions, median lesions below the elbow, plexus lesions which have not involved the lower trunk or medial cord, and all injuries below the head of the fibula, groups of painful and non-painful cases are made available for comparison in which the same nerve has been injured in the same region.

(v) *Significance of Preference for the Medial Cord of the Plexus, the Median Nerve and the Sciatic Nerve.*—The susceptibility to causalgia of the medial cord of the plexus and of the median and sciatic nerves is certainly connected with the fact that these trunks carry the bulk of the sensory fibres of the limbs. The sensitive palm and palmar aspects of the digits which are served by the median nerve and the medial cord of the plexus have proportionately many more sensory fibres than the rest of the skin of the upper extremity; they have also greater representation in the spinal ganglia and in the central nervous system. Similarly, the medial popliteal carries the bulk of the sensory fibres of the lower limb, most of which go to the sole.

Possibly there is some significance in the fact that a large proportion of the sympathetic fibres to the arm go to the hand; these travel in the medial cord of the plexus and pass to the median nerve by its inner head, while the bulk of the sympathetic fibres pass to the foot by the medial popliteal nerve.

2. *Level of the Injury.*

It has transpired already that the incidence of causalgia is greater in high lesions than in lower ones (Tables II and V). All 34 cases in this series followed lesions in the proximal segment of the limb. A similar disproportion has been reported by all other writers. Thus in Ulmer and Mayfield's series[24] 66 of the 75 patients have been wounded in the thigh or upper arm. Possible reasons for this will be discussed later. It has been

demonstrated many times that the ill effects of nerve injuries on the central nervous system are greater the more closely the level of injury approaches the nerve roots.

3. *Extent of Damage to Nerve Fibres.*

Percival Pott[32] stated that pain was more likely to follow partial than complete severance of a nerve. The amount of damage, however, is not always easy to assess, but will be examined in relation to such factors as the wounding mechanism, visible damage to the nerve, neuroma and scar formation, and the extent and quality of spontaneous recovery.

TABLE V.

Level of Median Lesions.

		Number of Cases.	Total.	Causalgia.	Total.
Upper arm	U/3	7	—	3	—
	M/3	10	—	2	—
	L/3	18	—	4	—
Total upper arm		—	35	—	9
Forearm and wrist		—	32	—	0
Total		—	67	—	9

U/3, M/3 and L/3=upper, middle and lower thirds of the upper arm respectively.

(i) *Wounding Mechanism.*—Causalgia is preeminently a result of battle casualties. Denmark's case resulted from a gunshot wound and so also did the series reported by Mitchell.

In the present series of 34 cases 30 resulted from gunshot wounds. In the remaining four the causes were:

Case 6: Fracture of pelvis involving sciatic nerve.
Case 20: Stretch injury of plexus.
Cases 32 and 33: Fracture of humerus involving radial nerve.

There was not one case of causalgia in the 30 nerve injuries due to lacerations available for reference in the full series. The reason for this lies, perhaps, not so much in the manner of infliction of the injury as in the regions favoured by this type of wound. Thus all 30 lacerations were in the upper limb; the median was involved in 14, only two of which were above the level of the elbow. If our previous conclusions relating to the identity of the nerve injured and to the level of the lesion are correct, it is clear that causalgia must be uncommon after this type of wounding mechanism.

The contrary was the case with gunshot wounds, the majority of which involved nerves in the proximal segment of the limbs (Tables I, II and V), and also with closed fractures and stretch injuries involving nerves which do not commonly occur in the distal segment.

The Stretch Factor.—An important feature of gunshot lesions of nerves is the factor of stretch. Puckett, Grundfest, McElroy and McMillen[33] recently demonstrated that a small high-velocity pellet passing through the soft tissues near a nerve will subject it momentarily to an astonishing degree of lateral displacement and stretch. In a battle injury of a nerve such a clean "through-and-through" wound by a small high-velocity missile probably indicates a greater degree of stretch than a larger wound with foreign bodies retained in the tissues. In this series 24 of the 30 gunshot lesions with causalgia were due to perforating high-velocity missiles; the patients in Ulmer and Mayfield's[30] series sustained high-velocity missile wounds.

In Cases 10 (double sciatic lesion) and 28 (double median lesion) severe pain followed the mild and transient lesions due to clean penetrating wounds, while painful sequelæ did not follow the large wounds with severe paralyses.

In Case 7, in which no recovery occurred, though the sciatic nerve appeared normal, we must presume that a segment of it has been subjected to a degree of stretch intense enough to rupture most of its axons and that the resultant attempts at regeneration were spread over a considerable length of nerve with the consequent production of diffuse endoneurial fibrosis rather than a discrete bulb. This tension factor probably accounts in part for those cases of causalgia occurring after stretch injuries of the plexus and after closed fractures.

The importance of the stretch factor will be emphasized further when the effects of nerve injuries on the central nervous system are under discussion. Many experiments have shown that the intensity of the retrograde reaction occurring in a neuron after severance of its axis cylinder is proportional to the degree of violence to which the axon is subjected.

(ii) *Visible Damage to Nerve.*—Early inspection has been practicable only when the nerve was laid bare in the wound or could be inspected during exploration. After a "near-miss" the nerve may appear normal and yet the stretch to which it has been subjected may result in the most severe and intractable of nerve lesions.

Data were obtainable in 13 of the 34 causalgic cases. In Case 4 the medial popliteal division of the sciatic nerve bore evidence of damage at 24 hours. In the remaining six sciatic lesions (Cases 1, 3, 7, 8, 11, 12) the nerve was inspected at intervals varying from three to 134 weeks after the wounding, and in none was there any macroscopic evidence of gross damage to the fibres. Of four median cases in which data were available, a neuroma was observed in three, this indicating the severance of an appreciable number of fibres. The remaining two cases were number 33 (radial involvement in fracture of the humerus with neuroma formation) and number 34 (ulnar causalgia with complete severance of the nerve).

Carter[21] attached importance to extraneurial and perineurial fibrosis as an ætiological factor, but his evidence is not convincing. Kirklin *et alii*[25] could find no correlation between the severity of the pain and the anatomical lesion found at exploration.

(iii) *Neuroma and Scar Formation.*—There is the possibility that the pain may proceed in some way from the naked regenerating fibrils in the proximal segment of the injured nerve. Doupe, Cullen and Chance[34] have speculated upon the possibility of the activation of these fibres by impulses in nearby sympathetic fibres. There are two clear reasons, however, why pain cannot be explained upon this basis: (i) The pain usually commences a few hours or days after the injury, before neuroma formation can have begun. (ii) Excision of the bulb is not often followed by cure and the operation has been abandoned as a standard procedure. A large number of authorities, commencing with Weir Mitchell,[29] could be cited to this effect.[12][14][30][32][34][35]

In Case 23—severe median causalgia which required sympathectomy at 30 weeks—the nerve appeared normal when explored, as also did the sciatic nerve in the six cases in which exploration was performed. There was no significant relation between neuroma formation and causalgic pain in this series. Tenderness in a neuroma should not be confused with causalgia. A nerve injury may be followed by the formation of a tender, palpable tumour of which the patient is at pains to take exquisite care at every moment of the day; yet there is no spontaneous pain. Speigel and Milowsky[32] found that

percussion at the site of injury did not precipitate or intensify causalgic pain; it appeared to them that the causalgic pain was not due to the continuous irritation of scar or foreign body. In Denmark's[1] case, however, "a small tumour could be felt in the site of the wound on the anterior part of the arm, which he could not bear to be *touched* without evincing additional torture".

(iv) *Degree of Recovery.*—In most of the cases the paralysis was complete at the outset, but the early appearance of recovery was considered as indicating that the lesion was of lessened severity. Late assessment of residual loss is the most reliable guide to the severity of the injury; 24 of the causalgia cases were followed up for more than two years and eight for more than four years.

TABLE VI.

Relative Severity of Lesions Based on the Onset and Progress of Recovery.

	Mild.	Severe.
Sciatic lesions:		
Non-painful	14	36
Causalgia	5	12
Lower plexus lesions:		
Non-painful	—	7
Causalgia	—	5
Median lesions above elbow:		
Non-painful	6	20
Causalgia	5	4

TABLE VII.

Relative Severity of Lesions Based on the Final Recovery.

	Poor.	Medium.	Good.
Sciatic lesions:			
Non-painful	17	9	24
Causalgia	2	5	10
Lower plexus lesions:			
Non-painful	2	1	4
Causalgia	1	2	2
Median lesions above elbow:			
Non-painful	2	8	16
Causalgia	—	2	5

In Table VI an attempt has been made to assess the severity of the lesion on the basis of the duration of complete loss of function and the speed of recovery, while in Table VII the degree of final recovery has been assessed into three grades—poor, medium and good.

(v) *Conclusions.*—It is clear that pain after nerve injuries is not confined to lesions of lesser intensity. The present series, however, was not large enough to decide whether or not the incidence of causalgia is greater after mild than after severe lesions. Speigel and Milowsky[23] found the nerve injury to vary from minor involvement to profound destruction, while others[24][25] have been unable to establish a correlation between the severity of the injury and the intensity of the pain.

4. *Damage to Non-Neural Tissues.*

(i) *Main Artery.*—There are three mechanisms by which damage to blood vessels may be thought responsible for pain in a limb.

(*a*) Ischæmia of Mechanical Origin.—Damage to a main artery may either occlude or severely reduce the blood supply to the limb, resulting in ischæmia with pain. The classical experiments of Lewis[36] demonstrated that muscular exercise under ischæmic conditions causes pain of a peculiar type; he further demonstrated[37] that the pain which follows arterial embolism is usually, perhaps always, due to muscular ischæmia and not to the state of the artery as such.

Ischæmic pain in muscle comes during exercise and eases when the muscle has rested. In causalgia this relationship does not apply; the pain sometimes is worsened or even precipitated by exercise, but does not subside with rest. Ischæmic pain has not a burning quality, nor is it commonly referred to the hand or the foot. It has been mentioned that aching pain may occur during exercise of a muscle weakened by deficient innervation. This does not happen, however, until the later stages, when the muscle is being reinnervated. On the other hand, the ischæmic muscular pain should be felt only in the earliest stages.

(*b*) Pain Radiating from a Vascular Lesion.—Leriche,[12] Temple Fay[7] and others have relieved severe post-traumatic pain in some cases by excising the damaged segment of an artery. Many careful observers have reported that arterial coats respond to needle puncture by giving rise to radiating

TABLE VIII.

Arterial Occlusion and Causalgia—Open Injuries.

Type of Lesion.	Number of Cases.	Occlusion of Main Artery.
Sciatic lesions :		
Non-painful	38	4
Causalgia	16	1
Median lesions above the elbow :		
Non-painful	26	14
Causalgia	9	3
Lower plexus lesions :		
Non-painful	5	—
Causalgia	4	1
Total :		
Non-painful	69	18
Causalgia	29	5

pain with a compelling quality and to other widespread reflex effects.[8][38] Myelinated and non-myelinated fibres have been demonstrated in the adventitial coats of arteries;[39] the myelinated fibres are little diminished by sympathectomy, but greatly reduced by posterior rhizotomy.[40] Though there are reports that arterial pain sense persists after division of sensory fibres to a limb[41] and is abolished, in some cases, by periarterial sympathectomy,[12][42] the evidence[8][43] indicates that this pain (i) is not diminished by sympathetic ganglionectomy and (ii) is subserved by somatic sensory fibres which temporarily accompany what have been described in macroscopic anatomy as periarterial sympathetic plexuses.

(*c*) Ischæmia of the Nerve.—Such a source of pain is unlikely, since anastomoses, on and within the nerve, between nutrient vessels derived from different and widely separated arteries, form the basis for the development of functionally effective collateral circulations when the major arterial channel to a limb has been interrupted. Collateral circulations of this type and their importance in maintaining the circulation of the limb have been reported on by various investigators.[44]

It might be thought possible, therefore, that damaged arterial tissue could give rise to spontaneous pain. Several recent writers,[16][20][25] however, could find no relation between causalgia and vascular damage, and the present series suggests that they are correct (Table VIII). The femoral artery was damaged in 4 out of 38 non-painful wounds of the sciatic nerve. In the causalgic cases the proportion was only 1 in 16; in that case the lesion was in the lower third of the thigh, where the nerve lies close to the artery. Sciatic causalgia, it will be remembered, is more common in the upper thigh, where the nerve is far from the artery. The median nerve, on the other hand, lies close to the brachial artery throughout its course in the upper arm, and as a result the latter is more often injured with the nerve. The vascular injury, however, appears to be without effect on the incidence of pain. In Case 23 resection of a traumatic brachial aneurysm gave no relief, but sympathectomy three weeks later effected a cure.

(ii) *Coexisting Bone Injury.*—Since causalgia does not commonly follow nerve lesions below the knee or elbow, the question of bone injury is practically limited to fractures of the femur and the humerus. Table I indicates the incidence of fractures in lesions of individual nerves and the relation of causalgia thereto.

Of 23 non-painful open injuries (21 due to gunshot wounds and two to other causes) of the median nerve in the upper arm, four were complicated by fractures—in the causalgia cases the proportion was 2 in 9. In the sciatic (all gunshot injuries) the proportions were 11:38 and 5:16. In the lower plexus lesions (all gunshot injuries) the proportions were 2:8 and 0:4.

In three cases causalgia followed nerve injuries which complicated closed fractures. The radial nerve was involved in Cases 32 and 33 and the sciatic in Case 6. The total number of lesions following closed fractures was only 16 (Table IX).

TABLE IX.

Nerve.	Number of Cases.	Causalgia.
Sciatic nerve	5	1
Brachial plexus	3	—
Median nerve	1	—
Radial nerve	5	2
Ulnar nerve	2	—

The time is not opportune for speculation as to whether or not these figures have general significance. Other observers[16][22] have reported that a concomitant bone injury was not a significant factor in their cases. It is possible, however, that the displacement of bone fragments may introduce a stretch factor or add to that generated by the missile in open injuries.

(iii) *Damage and Infection of Soft Tissues.*—Mitchell[2] thought that the onset of causalgia was often related to the appearance of "traumatic fever". Some have held that its incidence is favoured by severe damage or infection of soft tissues, while others have attempted to correlate it with the degree of surrounding fibrosis. These are variable factors and the means of assessing them are variable also. Often the observer has to guide him only the contemporary clinical notes and the amount of residual scarring, and it is difficult to separate the factor of tissue damage from the factor of infection. For present purposes these factors have been graded roughly into only two categories: (1) significant, (2) not significant (Table X).

Extensive injuries may be painful for a time, especially if badly infected and if much handling is necessary, while the final non-neural defects which result from scarring may be the cause of aching pain on exercise or of discomforts due to limitation of movement. Table X indicates generally that there is no constant relation between causalgia and damage to, or infection of, soft tissues; this is in agreement with the findings of other observers.[21][22][24][35]

TABLE X.

Causalgia as Related to Soft Tissue Damage and Infection Following Open Injuries.

Nerve and Type of Lesion.	Number of Cases.	Significant.	Not Significant.
Sciatic:			
Non-painful	38	25	13
Causalgia	16	10	6
Lower plexus:			
Non-painful	5	3	2
Causalgia	4	2	2
Median above the elbow:			
Non-painful	23	15	8
Causalgia	9	3	6
Total:			
Non-painful	66	43	23
Causalgia	29	15	14

In both the cases of bilateral lesions—Cases 10 (sciatic) and 28 (median)—pain followed a transient paralysis due to a clean perforating wound, while severe damage to nerve and soft tissues on the other side was not accompanied by pain.

5. *Trophic Changes.*

It has been known for many years that denervation of tissues results in atrophy; that muscle fibres waste away in a state of uncontrolled fibrillation; that squamous epithelium becomes thinned and shiny; that subcutaneous fascia withers, and fatty tissues lose their structure. Samuel[45] postulated the existence of trophic nerve cells whose function was to superintend the nutrition of the tissues. Sherrington[46] pointed out, however, that the normal nerve supply to a tissue is its trophic nerve supply: "Every nerve and nerve centre that can influence the proper and specific activity of a tissue—and that is the sole function of nerves—possesses, in so far, a trophic influence on that tissue. If by trophic nerves be understood no more than this, all nerves are trophic nerves."

Paget[29] and Mitchell[2] stated that causalgic pain was always accompanied by "glossy skin" on the palm (or sole) and on the digits; they suggested that trophic wasting was a special feature of these cases. In some of the present series trophic changes were profound, with ulcers on the fingers or on the sole. These sequelæ, however, were seen with equal frequency in non-painful cases. In the palm and sole the changes that accompany denervation are more manifest because of the special qualities of the epithelium, which thins away, and in the fingers the terminal pads atrophy, giving a tapering appearance especially to the index finger (Figure 1). These effects, of course, are seen more after lesions of the median and the medial popliteal nerves, which provide the main nerve supply to the susceptible areas. Causalgia, as it happens, is confined chiefly to the median and medial popliteal nerves, and if trophic changes are prominent it is because the hand has been denervated, not because it is painful.

In the present series the degree of trophic change appeared to be related to the degree of denervation; that is, trophic change always was greater in complete severance of the median than in partial loss. In Case 28 there was no trophic change on the left (painful) side, where the median lesion was not severe; but the changes were gross on the right, where the lesion was severe but not followed by pain (Figure I).

6. *Psychic Factors.*

"Perhaps few persons who are not physicians can realize the influence which long-continued and unendurable pain may have upon both body and mind . . . under such torments the temper changes, the most amiable grow irritable, the soldier becomes a coward, and the strongest man is scarcely less nervous than the most hysterical girl" (Mitchell[2]). Harassed by continuous pain and lack of sleep, the patient after a time may become a nervous wreck. If seen at this stage by an enthusiastic clinician with a psychosomatic bias, he may have his mind probed in a search for "psychic trauma". And the practitioner who suspects a psychogenic cause does not often fail to uncover a confirmatory history. Some physicians and surgeons, finding it difficult to appreciate the fact that these patients suffer intensely, are ever on the alert for evidence which may suggest that they are dramatizing or exaggerating their symptoms. To adopt such an attitude is in fact to commit a grievous error; apart from the serious mistake in diagnosis, a great injustice is being done to the patient in attempting to lay upon him both the blame for being ill and the responsibility for curing himself. The patient is the only witness we have, and if he says he has a pain there are no grounds on which we can contradict him.

Many who try to explain such pains upon a psychogenic basis seem to have made no clear distinction between the perception of pain that is present and the imagining of pain that is not there. There is, of course, a cortical element in every act of perceiving a pain, even as there is a cortical element in perceiving a sight or a sound. But the fact that the observer cannot personally confirm the patient's act of perception (as he can confirm the sound that he also hears, which the patient describes) sometimes causes him to regard the pain as somehow less real than other more material perceptions, and to imagine that it can be relieved by the patient's mental efforts. Lidz and Payne[17] recently reported relief of a causalgic type of pain by hypnotic suggestion. Such a result, however, in a suitable subject does not negative the reality of the pain.

If the practitioner begins to have doubts about the reality of the patient's sufferings, it is not likely that he will be able to hide these doubts from the patient. He is bound to betray himself in some way to the hypersensitive subject or he may deliberately express his doubts in a well-meant explanatory essay at psychotherapy. The result will be that to the patient's physical sufferings will be added the mental torment of knowing himself disbelieved.

Some writers adduce, as evidence for the psychic nature of causalgia, the fact that exacerbations of the pain may be brought on by mental exhaustion or by visual, auditory or psychic stimuli. In Case 29 the patient said, some two years after his injury, that a scraping sound could bring on a flash of pain in his arm; and the patient in Case 28, after one and a half years, felt pain in his arm when he saw two cars narrowly avert a collision. Such phenomena can be more easily comprehended, however, if, as Livingston[30] suggests, the sensory nervous system is considered as a pool of neurons into which flow stimuli from all parts of the body. In causalgia the spontaneous activity of the part of the pool which subserves pain is greater than normal,

and normal impulses flowing in may summate to raise this activity into the level of the consciousness when it is felt as pain. "Excitation in a pool of neurons", said Gasser,[48] "is dependent upon everything which is happening in the nervous system"; and a shower of stimuli from cortical levels may be as effective sometimes in causing this pool to overflow as is a shower of stimuli from somatic levels.

7. *Indications of Sympathetic Involvement.*

(i) *The Afferent Functions of the Sympathetic.*—Painful responses to mechanical stimuli are not diminished by sympathectomy, but evidence has been accumulating which suggests that afferent impulses carried by anatomically described sympathetic trunks (as defined by dissection) sometimes play a part in the production of spontaneous pain [see Section V, 4, (*b*)]. Slaughter[49] employed sympathectomy to relieve pain in the lower limbs of a patient whose cord had been completely transected at the first lumbar segment. He argued that this patient must have had somatic afferent fibres travelling in his lumbar sympathetic chain. These deductions greatly interested Hyndman and Wolkin[50] and they proceeded to make some interesting observations upon patients who had been subjected to unilateral sympathectomy. Upon exposure of the whole body to a low temperature, a normal patient complains of a general sensation of cold, with stinging pain in the ears and the fingers and toes. The patients who had been subjected to sympathectomy, however, observed that the pain was not felt on the operated side, where the extremity gave a subjective sensation of warmth, though objectively it became as cold as the normal limb. Furthermore, in two of their cases "the lower extremities were supplied by sympathetic fibers but were devoid of somatic innervation". Sensation could still be evoked by appropriate stimuli. Recently Kuntz and Saccomano[51] demonstrated, in experiments upon cats whose cords had been transected in the upper lumbar region, that painful stimuli to the hind legs caused reflex dilatation of the pupil. This response was abolished by sympathectomy.

(ii) *Sweating and Flushing of the Extremity.*—Twelve of the 34 causalgic patients claimed that the extremity sweated more than its fellow. In the remainder no observations were recorded. The patient in Case 28 (bilateral median lesion) states, six years after injury, that the left hand (mild injury, causalgia) still sweats more than the right (severe lesion). But the paralysed hand of the patient in Case 20 (plexus stretch lesion with Horner's syndrome) was completely dry. André-Thomas[30] reported a case of gunshot wound of the plexus with causalgia and complete sympathetic paralysis for three months. After that time the sweating function returned to normal. Mitchell[2] observed that partially denervated skin often sweated excessively whether painful or not; our observations confirm this.

The effects of denervation upon the circulation of an extremity are variable, depending upon the trophic state of the tissues, the degree of denervation and the amount of regeneration. The flushing which follows sympathectomy is of short duration and is due to the temporary abolition of vasoconstriction. Under some circumstances the sympathetic can bring about active vasodilatation,[52] but this is also known to be an "antidromic" response. In five cases (18, 19, 25, 26, 29) flushing of the painful hand was mentioned. The phenomenon was not recorded in any of the painful feet, probably because it was not seen, the feet being under cover for most of the time. However, as Doupe, Cullen and Chance[34] have already pointed out, "numerous interpretations could be placed on the finding of an increased flow in an injured extremity", and these are increased if there is a coexisting nerve injury. In all of White's[35] 11 cases, however, the affected extremity

was colder than its fellow. Other observers have reported variable sympathetic effects.[10][23][24]

Neither from the disturbances of sweat secretion, however, nor from alterations in circulation, is any evidence obtained which certainly implicates the sympathetic as a factor in causalgia.

(iii) *The Results of Sympathectomy.*—Leriche[30] claimed good results for "arteriectomy" when persistent pain followed vascular injuries, and later for "periarterial sympathectomy" for painful cases in which the vessel had not been damaged. At a later stage section of the sympathetic fibres, either on the distal or the proximal side of the ganglion, replaced the arterial operation, which has no effect on the sympathetic supply to the vessel below that level. Sympathetic block by local anæsthesia was found to abate the pain temporarily and in a few cases complete relief was reported.[12][14][16][22][34][35][54] In the present series this procedure was used only in Case 11 (sciatic lesion), in the early records of which occurs the statement that lumbar sympathetic block relieved the patient's pain for two days.

A number of writers have reported good results from sympathectomy in causalgia.[10][12][21][24][25][28][29][34][35][54][55] The preganglionic is preferred to the post-ganglionic operation. Rasmussen and Freedman,[10] Kirklin *et alii,*[25] and White[55] found it decidedly more reliable.

The occasional necessity for repeated operations involving wider and wider excisions of ganglia has been recently reported. Thus de Takats[56] and Ulmer and Mayfield[29] sometimes find it necessary, for pain in the leg, to excise ganglia as high as the eleventh thoracic segment.

In five sciatic and two median injuries of the present series preganglionic sympathectomy was performed by Mr. Hugh Trumble. The operation failed in one sciatic injury (Case 1); it had been delayed for 13 months. In Case 2 there was no improvement for a few days after the operation, and it is doubtful whether the ultimate success can be attributed to the sympathectomy. In all of the others the operation was succeeded by marked relief, though in Cases 3, 4 and 5 the patients continued to have occasional attacks of very mild pain. In Case 24 the patient was suffering so acutely that the operation was performed as an emergency at night; when he awoke from the anæsthesia the pain had disappeared. In some of the other cases operation was thought necessary at one stage, but after a short delay it became obvious that a decline in the severity of the pain had commenced. In none of the cases in which improvement occurred after sympathectomy did the hyperalgesia diminish perceptibly. As in the case of Kirklin *et alii,*[25] it has not been possible to establish any correlation between the result of the sympathectomy and either the type of pain or the duration of the interval elapsing between injury and the operation.

In assessing the results of operations for pain it is wise to maintain a conservative attitude and to remember that, while excellent results appear to follow sympathectomy today, equally excellent results were claimed in the past for procedures which later were abandoned. At the moment the evidence suggests that, of the extraspinal procedures, preganglionic sympathectomy is more likely to bring relief than any other measure.

The role of the sympathetic will be considered further in a subsequent section.

VI. ANATOMICAL AND PHYSIOLOGICAL CONSIDERATIONS

1. *Pain Pathways.*

Much evidence indicates that the fibres which convey pain are mostly non-myelinated or very finely myelinated and that they spring from the small unipolar cells of the spinal ganglia.[57][58] The peripheral fibres of these small

cells branch in nerve trunks, as well as at the periphery, so that the same ganglion cell may serve a wide territory.[59] Their central processes, which comprise the lateral division of the posterior roots, branch before making contact with the cells of the *substantia gelatinosa* of the same and neighbouring segments.[60] The central neurons are so linked that impulses from one cell may affect a large number of its fellows while pain messages may be relayed several times and follow intricate paths in the posterior horn of grey matter before finally ascending in the spinothalamic tract of the opposite side. May[61] states that some of the ascending fibres terminate in the intermediate grey matter and "by means of a series of short chains, afford secondary paths to the same end station, which may supplement the direct path, or be made available after interruption of the direct path". There is also experimental evidence that pain from one side may ascend on both sides of the cord.[62][63]

Electrophysiological research indicates that sensory messages are relayed through an internuncial neuronal pool in the posterior horn, whose cells are so linked as to provide a great variety of pathways through which an impulse may travel.[64] Some of these linkages are "closed chains" in which the neurons may reexcite each other for prolonged periods. The arrival of an afferent impulse thus results in prolonged activity in the internuncial pool, which manifests itself as a sustained barrage involving several segments, while antidromic impulses can be recorded in the posterior roots of the same and adjacent segments and on the opposite side.[65] Most of this evidence relates to large myelinated fibres, and how much, if any, of it is applicable to the pain fibres remains to be demonstrated. It is possible, however, that the latter system will behave like the larger units, whose reaction can be observed. If this be so we must assume that the continuous and widespread activity occurring in the cord is so integrated under normal conditions that it produces no abnormal effects outside the cord either in the centres of consciousness or in the peripheral tissues. If for any reason it were to become disordered at a given spot, wider and wider areas of the cord may gradually become involved in a spreading disturbance, producing effects both on the sensorium (spontaneous pain) and on the peripheral tissues (hyperalgesia). This is the basis of Livingston's hypothesis[14] that causalgia is the expression of "an abnormal state of activity in the internuncial neuron centers of the spinal cord gray matter" created by impulses from "an organic lesion at the periphery involving sensory filaments".

2. *Changes Occurring in Neural Pathways after Injury.*

The Effect on the Cell Body.—Section of a peripheral nerve causes changes in the parent neurons which are usually visible within seven days. The reaction is characterized by swelling of the cell, dissolution of the Nissl granules, vacuolation of the protoplasm and dislocation of the nucleus. The final result is either (*a*) the death of the cell or (*b*) its return to normal within 40 to 90 days. Observations on human and experimental material indicate that the intensity of the reaction: (i) Varies in different species of animals and even from case to case in the same species.[66] (ii) Is proportional to the violence done to the fibres.[67][68] Marinesco[67] used graded degrees of violence in stretching nerves and found the reaction to be proportionate to the violence. (iii) Is inversely proportional to the distance of the lesions from the spinal ganglion.[66][68] Thus the reaction is greater following proximal than distal lesions. (iv) Is often greater in sensory than in motor neurons and more especially in the small cells of the spinal ganglia.[57][67][69][70][71] Irritation superadded to the trauma accelerates the changes, the small cells participating to a greater extent than when there is no irritation.[69a]

Marinesco[67] refers to Brown-Sequard and other French physiologists who were unable to explain the disproportionate reaction in the sensory cells after a lesion in a mixed nerve. They concluded, erroneously, that the sensory fibres must be situated peripherally where they would presumably suffer the most violence.

Section of sympathetic fibres is also followed by a retrograde reaction in the parent cells.[72]

Transneuronal Degeneration.—Transneuronal degeneration is commonly observed in experimental neurology and probably plays a large part in neuropathology.[72]

The collaterals of the posterior root fibres branch freely before ending in contact with the neurons of the posterior horn of grey matter. If a sensory ganglion cell perishes as the result of a peripheral nerve injury, these collaterals will also degenerate. This is followed by changes, on both sides of the grey matter of the cord, in the cells with which they had communicated. Transneuronal degeneration of the cells in Clarke's column which have been partially or wholly bereft of synaptic contacts has been frequently observed.[74] These activities, however, can be followed only in the larger collaterals, which deal mostly with proprioceptor impulses. Less is known about degeneration in the smallest fibres carrying pain impulses, while their connexions are also small and difficult to demonstrate. Following nerve section, however, the casualty rate among small ganglion cells is four times as great as among large neurons,[57] while a severe loss of both cells and fibres in the *substantia gelatinosa* has also been observed.[75] For these reasons transneuronal changes should be expected on the pain pathway following peripheral nerve section.

Bilateral Central Effects.—The contralateral spread of the effects of nerve injuries has been observed experimentally.[71][74][76] This evidence provides a basis for the bilateral spread of sensory disorders. Though no case of contralateral spread of the pain has been reported in the present investigation, such cases are common in the literature.[2][12][14][28][77][78][79]

3. *Antidromic Effects.*

Bidirectional conduction in nerves and across junctions is now well established.[2][8][65][80] Most of the early studies of antidromic impulses were concentrated upon hyperæmia, but Lewis[8] later demonstrated that hyperalgesia is also an antidromic effect in which a substance is liberated in the tissues which sensitizes the pain receptors. In the "minor causalgias" several investigators have suggested that the hyperalgesia, hyperæmia and decalcification are reflex antidromic effects.[12][14][15] When Foerster[81] stimulated the distal part of a cut nerve root the patient felt pain in the margins of the cutaneous field of the root. When he cut the neighbouring roots on either side this pain was no longer felt. This suggested that the pain had been the result of a change brought about in the tissues by the antidromic impulses and recorded by the receptors connected with neighbouring roots. More recently, Pool and Brabson,[82] when exploring severed injured radial median or ulnar nerves under local anæsthesia, electrically stimulated the distal stump; the patient invariably felt pain in the distribution of the nerve. After blocking of the intact sensory nerves the pain was no longer felt.

If it be true that impulses flow outwards through the posterior roots, then continuous disorderly activity in the internuncial pool of the cord will make its presence felt by a continuous efferent discharge through a large number of roots. This may account for the hyperalgesia in causalgia, which varies in extent and intensity from day to day and often spreads beyond the field of the injured nerve.

VII. THE RESULTS OF SURGICAL TREATMENT FOR PAIN.

Every case of injury to a nerve, or of a surgical operation upon a nerve, should provide opportunities for making important observations. The long history of the changing surgical attacks on pain has provided experimental evidence of a valuable kind and from their results it should be possible to draw deductions as to the level at which the pain is generated.

Nerve Block, Neurolysis, Neurotomy and Amputation.—Injury to a nerve trunk sends off a shower of abnormal impulses[90] and, since these travel in either direction, both peripheral and central effects could result. But the pain fibres in a nerve trunk are not especially susceptible to lesser mechanical stimuli such as compression of medium degree. Nerve trunks are tender to heavy pressure, but the tenderness is located at the site of compression rather than in the distribution of the nerve. The observer can confirm this by compressing his own ulnar nerve; he can observe also that light pressure, if prolonged, will interrupt conduction without pain. Crutch palsy and plaster compression palsy of the lateral popliteal nerve at the neck of the fibula are examples of painless compression of a nerve. This was well known to Waller[84] and to Head,[85] who wrote, "pressure on nerve trunks causes numbness and tingling, but not superficial tenderness". Pain is not a prominent feature of tumours of nerves, malignant or benign,[86] and apart from causalgia it has not been conspicuous in the present total series of nerve injuries. In Speigel and Milowsky's[22] series the pain was not precipitated or intensified by percussion at the site of injury. In Case 27 in this series, however, a slight blow on the wound would precipitate an attack of pain in the hand, while touching the "tumour" at the site of injury in Denmark's case greatly accentuated the pain.

Boring,[4] Sharpey-Schafer[5] and Trotter and Davies,[87] who submitted themselves to section of sensory nerves, felt spontaneous pain in the denervated area. Schafer considered the possibility that the pain and accompanying hypersensitivity might be due "to changes occurring in the sensory ganglion cells". By pressing on the ulnar nerve (which supplied the severed branch in his experiments) he was able to prevent the passage of impulses along its fibres. He observed that "while the compression lasted the pain ceased to be felt; as soon as the pressure was released and the nerve fibres had recovered from it, the pain reappeared." He therefore concluded that the cause was peripheral.

The history of alcohol injection, of the injection of various solutions either as local anæsthetics or designed to disrupt suspected intraneural fibrosis, of nerve ligation and of nerve crushing, has been one of repeated failures. Occasionally relief is obtained by freeing the nerve from adherent scar tissue. In Case 8 the patient claimed that there was a great improvement after neurolysis at 134 weeks, and in Case 12 the patient thought that a like procedure brought an end to his pain at 47 weeks. Neurolysis, however, is usually unsuccessful.[29][32][34][35]

Denmark[11] claimed that he had effected a cure in his case by amputation. Earle[31] a few years later reported the cure of a case of severe ulnar neuralgia by neurotomy. Weir Mitchell,[88] however, could not overlook the general failure of neurotomy, which, he considered, occurred only because it was delayed too long, allowing the spread of an "ascending neuritis". Carter[31] was the last writer to advise neurotomy seriously as a routine procedure; others have reported only occasional successes.[14][34][35] Leriche[12] was opposed to it.

Tinel[89] reported a case of causalgia which had been cured by section of the nerve trunk distal to the lesion. This was interpreted by Lewis[8][90] as indicating that the pain depends upon efferent impulses through a damaged,

but not completely interrupted, nerve trunk. Tinel's experience has received limited corroboration from Livingston[14] who was able, by anæsthetizing the ulnar nerve at the elbow, to relieve, for the duration of the analgesia, causalgic pain from an ulnar lesion at the mid-humeral level. Neurotomy, however, completely relieved the patient for only a few weeks, when the pain returned with all its previous intensity. In three of the causalgia cases reported by Doupe, Cullen and Chance[34] relief was obtained by nerve block below the lesion, but this procedure failed in another case. De Takats[64] recently said that causalgia is not a sympathetic dystrophy but "a true antidromic nerve stimulation". The weight of the evidence, however, indicates that the peripheral receptors play a secondary part only. The pain of a phantom limb often closely resembles causalgia; here the peripheral receptors are absent.

Though some observations suggest that the site of damage may provide the discharging focus or "trigger point" in causalgia, this is unusual. The weight of evidence indicates that in causalgic pain the responsible factor is situated further centrally in the cord and that, though in the initial stages peripheral damage is responsible for inducing the central changes which initiate the disorganization of cord activity, these may become so established that the central focus continues to function abnormally in the absence of impulses arising from the site of damage.

Posterior Rhizotomy.—Bennett[91] was the first to report the operation of posterior rhizotomy for severe neuralgic pain; his patient died ten days after the operation. Abbe[92] reported three cases in which the persistence of the pain led him to believe that the patients were imagining their symptoms. The case reported by Morton Prince[93] was no more successful and the history of posterior rhizotomy for pain is one of repeated failure.[94]

Spinothalamic Tractotomy.—Often the relief of spontaneous pain is not permanent, the pain recurring on the same or on the opposite side.[14][62][79] In many cases of pain in the lower limb or in the trunk, however, chordotomy has proved a valuable palliative method,[35] and Falconer and Lindsay[96] have reported the successful treatment by chordotomy of painful phantom hands and causalgia. More recently, notable successes have been achieved by cutting the tract in the medulla[96] and in the mid-brain.[97] The occasional impermanence of the results of spinothalamic tractotomy indicates that alternative paths may be available.

Ablation of the Sensory Cortex.—Mahoney[98] reported the cure of phantom limb pain by removal of the relevant area in the sensory cortex. In the hands of others, however, the relief has been but temporary.[99]

Sympathectomy.—The relief obtained in causalgia by preganglionic sympathectomy has yet to be explained (see previous section). Most of the explanations given are based on the assumption that the sympathetic outflow has a sensitizing effect on the pain receptors in an already hypersensitive limb. But they fail to take into account the fact that amputation and high neurotomy, both of which interrupt these sympathetic fibres, have been discredited because they so frequently fail to relieve the pain. It is surprising, therefore, to find that success follows section of the sympathetic fibres alone. It may be that the operation gives relief because the interruption of sympathetic tracts modifies, in an as yet unexplained way, the disordered pattern of internuncial activity in the cord.

Doupe, Cullen and Chance[34] attribute the causalgic pain to disturbances created in sensory fibres at the site of injury by efferent impulses in adjacent sympathetic fibres. They marshalled some interesting observations in support of their claim, but the repeated failure, in many hands, of high and low

neurotomy and nerve block and the rapidity of onset of pain in so many cases render such an explanation untenable. Furthermore, in some of their cases they were forced to postulate a conjunction of sympathetic and sensory fibres at their termination at the periphery for which, as they themselves admit, there is not a shred of evidence.

We should remember that many procedures for the relief of pain have had their crowded hour of general enthusiastic adoption only to fade gradually into oblivion. Results were claimed at first for neurotomy, amputation and posterior rhizotomy, which experience could not confirm. In succession "periarterial" and postganglionic sympathectomy enjoyed each its vogue of acclaim, only to be replaced by preganglionic sympathectomy, and here we find that, on occasion, it is necessary to do repeated operations and progressively wider excisions. We cannot claim that sympathectomy is of universal value, as we can claim that chordotomy at a higher level certainly interrupts the chief pain pathways.

Conclusions.—The clinical evidence suggests that causalgia is the effect of the spread of the damage to the central nervous system and that pain, even though localized, often indicates a disturbance of an area of grey matter much larger than that which its distribution appears to represent.

Operations have been performed for pain at nearly every possible site in the pathway from the peripheral receptors to the sensory cortex, and at every level the story is the same—some encouraging results but a disheartening tendency for the pain to recur. At whatever level pain is attacked, the impression is conveyed that the whole nervous system makes a coordinated effort to reestablish the pathway. Livingston[44] was impressed "with its remarkable ability to find a new route when the customary channels have been blocked". Leriche[129] was also discouraged by the failure of surgical attacks to relieve pain: "nerves are not made to be divided, a demonstration . . . which surgery was long in discovering, and which is not even yet universally admitted as an established fact" (page 215).

Repeated section of peripheral nerve fibres means in effect the repeated section of axis cylinders of surviving sensory cells, with the death of some and a spread of disorganization amongst internuncial neurons. Rhizotomy means the degeneration of the central fibres, with transneuronal effects upon the grey matter. Those who do large numbers of antero-lateral chordotomies usually give evidence of diminishing conservatism; increasingly wide sweeps of the knife are advocated in order to be certain of interrupting all the pain fibres. Sometimes bilateral chordotomy is advised.[28][70][90][100]

The mechanism of the relief often given by sympathectomy can only be guessed at. We are left with the surmise that preganglionic sympathectomy frequently relieves causalgia because the changes which this procedure induces influence the conditions which have been assisting in the perpetuation of a state of disorderly activity in the sensory neuronal pool in the cord.

VIII. DISCUSSION: AN EXLANATION OF THE CAUSALGIC STATE.

The facts which emerged from the earlier study of causalgia can be related to the intensity of the retrograde reaction which follows nerve damage. As we have seen, the latter is guided by three main factors: (i) The reaction is more intense in sensory than in motor neurons, and more in the small (pain) cells than the large ones. (ii) Its intensity is inversely proportional to the distance of the injury from the spinal ganglion. (iii) It varies with the amount of violence done to the nerve.

As we have also seen, the incidence of causalgia is affected by three factors: (i) It follows damage to the main sensory paths to the hand and the foot. The median field has a far greater sensory representation in the

central nervous system than any other nerve in the arm and makes contact with internuncial neurons in large regions of the cord. The same remarks apply to the medial popliteal. The retrograde and transneuronal effects of damage to these nerves should therefore be proportionately great. (ii) Causalgia more commonly follows high than low injuries. (iii) It appears to be related to the amount of stretching violence to the nerve.

Causalgia, however, is not a manifestation only of chromatolysis in spinal ganglia, for, if it were, rhizotomy would cure it. Usually, however, the pain abates after this procedure only to return after a few weeks.[12][14] The clinical evidence suggests that the painful sequelæ of nerve injuries are the effect of the spread of the damage to the central nervous system, and morphological and physiological evidence lend support to this interpretation.

Distress or death of the sensory ganglion cells induces a reaction in the central cells with which it is connected. It is tempting to postulate that this transneuronal impairment of function, which may proceed to degeneration, is manifested as abnormal spontaneous activity in the neurons of the posterior horn of grey matter. As a result wider and wider areas of the cord may, even in the absence of a peripheral trigger point, gradually become involved in a spreading disturbance, producing effects on the sensorium (spontaneous pain) and on the peripheral tissues via adjacent posterior roots (hyperalgesia). If, as all of these considerations indicate, causalgia depends upon a disordered pattern of activity in the cord which sends a succession of abnormal impulses to the cerebrum, normal impulses from different parts could conceivably summate to raise this activity above the threshold and pain would result or be aggravated. Such a conception is supported by clinical observation, which has shown that a great variety of stimuli may increase the pain or precipitate attacks (see earlier section relating to the factors responsible for the exacerbation of pain). However, while extraspinal influences play their part in precipitating or perpetuating this activity, the essential feature is the disorganized intraspinal pattern.

As the central cellular reaction in response to peripheral damage settles down and readjustment takes place, these effects (pain and hyperalgesia) may diminish or disappear, thereby accounting for the cases which undergo a spontaneous cure. An instructive case was recently reported by Moore,[101] who, before operating for hernia, gave a spinal anæsthetic to a patient whose leg had been amputated 29 years before. For the duration of the anæsthesia he complained of pains in the stump so severe that he cried out in agony. When feeling returned to the limb remnant the pain abated and did not return. It would seem that in this patient an artificial balance had been achieved between the depleted primary and internuncial neurons of the damaged limb. Removal of extraspinal influences left the internuncial neurons in a state of disorganization for which they were unprepared, and the result was the dispatch upwards of an abnormal succession of impulses.

The anatomy of pain pathways, the central changes induced in them by peripheral damage, electrophysiological observations on cord activity and clinical observations on the painful sequelæ of nerve injuries all support Livingston's postulate that causalgia is a manifestation of "an abnormal state of activity in the internuncial neuron centers of the spinal cord gray matter" created by impulses from "an organic lesion at the periphery involving sensory filaments". Such an explanation obviously fails to account for the absence of pain in the majority of peripheral nerve injuries in which the damage should, on the basis of the argument set out, have induced central changes of the type held responsible for causalgia. The present state of knowledge regarding these central effects, however, is so fragmentary that it can provide only a guide to the structural and functional changes

responsible for the condition. The apparent inconsistencies will be explained only when further and more extensive investigations reveal details of the obscure processes involved.

No evidence has emerged from this study to suggest that causalgia is in any special sense a disorder of the sympathetic nervous system. The relief obtained by preganglionic sympathectomy has yet to be explained. It may be that the operation gives relief because the interruption of sympathetic tracts modifies, in an as yet unexplained way, the disordered pattern of cord activity.

Treatment.—In most cases causalgia undergoes a spontaneous cure and surgery should therefore be reserved for the severe and stubborn cases. The conservative treatment of patients does not call for comment.

The paths by which pain impulses travel appear to be tortuous; that they become more so when a dynamic disorder of sensory neurons has commenced is suggested by the conflicting results of surgical attacks on pain pathways. Thus even the most skilfully planned operation to interrupt the pathways may fail to cure the pain.

Since many cases of causalgia proceed to spontaneous recovery which is either complete or of a very high order, any procedure which would jeopardize such recovery is unjustified. If any attack on the peripheral nerve is contemplated this should be confined to neurolysis or the injection of non-sclerosing anæsthetic agents. But experience has shown that there will be few occasions when adherent scar tissue will be the agent responsible for severe and prolonged pain and neurolysis prove a relieving measure, while few successes can be anticipated from the injection of anæsthetic agents. A neuroma should be excised only if there is good reason for believing that it is hindering regeneration.

The foregoing study indicates that there is a high incidence of success following preganglionic sympathectomy. When this procedure fails, chordotomy will account for all but a few of those cases in which the pain persists.

SUMMARY.

1. The paper reports the results of an investigation of the painful sequelæ of injuries to peripheral nerves. Observations were based on cases in which severe pain in the hand or foot had persisted for at least five weeks after the receipt of an injury to a nerve trunk and in which the pain was not related to damage or to involvement of non-neural tissues. The term causalgia has been applied to this pain.

2. Causalgia is primarily a sequel of gunshot lesions in which stretch of the nerve appears to play an important part.

3. In the great majority of cases it follows damage to the main sensory pathways from the palm or the sole.

4. The higher in this pathway the level of the lesion, the more likely is causalgia to ensue.

5. Its incidence bears no relation either to neuroma formation or to the degree of visible damage to nerves or other tissues.

6. Though sympathectomy has proved a valuable therapeutic measure there is no other evidence to suggest that the sympathetic system is especially involved.

7. The clinical evidence suggests that the painful sequelæ of nerve injuries are the effect of the spread of damage to the central nervous system by retrograde and transneuronal changes. A centre of abnormal spontaneous activity is thereby set up in the cord, which acts as a focal point for the dissemination of spreading disturbances involving wider and wider areas

of the cord. These produce effects on the peripheral tissues via adjacent posterior roots (hyperalgesia), while a succession of abnormal impulses reaches the cerebrum, producing effects on the sensorium (spontaneous pain). Anatomical and physiological evidence is produced which lends support to this interpretation.

8. The results of the surgical treatment of pain have been reviewed in relation to the anatomy and physiology of pain pathways and to the postulated focus of disturbance in the cord following peripheral nerve injury.

9. Most cases of causalgia undergo spontaneous cure and surgery should therefore be reserved for the severe and stubborn cases. The paths by which pain impulses travel are tortuous; and they may become more so when a dynamic disorder of sensory neurons has commenced. Thus even the most skilfully planned operation to interrupt the pathways may fail to cure the pain. At the moment the evidence suggests that, of the extraspinal procedures, preganglionic sympathectomy is more likely to bring relief than any other measure.

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THE END RESULTS OF OPERATIONS FOR INTRATHECAL EXTRAMEDULLARY SPINAL TUMOURS, WITH A REPORT OF NINE PATIENTS WHOSE TUMOURS WERE REMOVED BETWEEN 14 AND 22 YEARS AGO.[1]

By LAMBERT ROGERS,
Professor of Surgery, University of Wales.

THE surgery of spinal cord tumours may be said to have begun on June 9, 1887, when Sir Victor Horsley at Queen Square removed one from within the meninges of the upper thoracic region of a paraplegic and incontinent army officer, aged forty-two years, who made so complete a recovery that a year later he was hunting. He remained well up to the time of his death from another cause some twenty years later. The tumour was described as a myxoma that was in part cystic, which suggests that nowadays we should probably classify it as a neurinoma. Unfortunately this is not capable of verification, as it was destroyed on May 10, 1941, along with other specimens in the museum, when the Royal College of Surgeons of England received a direct hit from a high-explosive bomb dropped from an enemy aircraft.

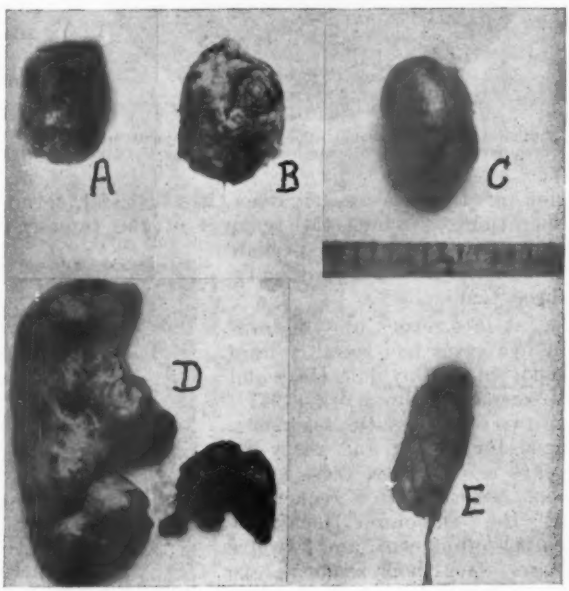

FIGURE I. Some spinal tumours from the writer's collection, removed between the years 1929 and 1934. *A* from the upper thoracic region of a woman, aged 42 years; *B* from the lower thoracic region of a man, aged 36 years; *C* from the upper thoracic region of a man, aged 33 years; *D* from the upper cervical region of a man, aged 23 years; *E* from the mid-thoracic region of a man, aged 32 years. *A* and *B* are meningiomata. *C*, *D*, *E*, neurinomata.

It is one of the most satisfying experiences given to surgeons to see a patient cured of the palsy; to see one who is paralysed and incontinent regain the use of his limbs and control over his sphincters, and in fact "take up his bed and walk".

Elsewhere (Rogers, 1932) I have discussed the factors which appear to influence the degree of this often astonishing restoration of function. We

[1] From the Surgical Unit, the Welsh National School of Medicine, the Royal Infirmary, Cardiff. Accepted for publication on May 17, 1948.

may inquire, however, whether such gratifying recoveries always persist over the course of years or whether relapse is liable to take place, either from local recurrence of the tumour or from complications following the operation. Cushing and Eisenhardt (1938) state that the end results are, generally speaking, most favourable. Ten of their eleven patients with neurinomata made good recoveries and nine of these patients were living and well, with no evidence of recurrence, from six to twenty-eight years later. The results were not quite so pleasing in the case of the meningiomata. In their series of 17 verified examples there were three operations for recurrence — after two years in each of two cases and after nine years in the remaining case, which had a fatal termination. In two other cases death had occurred from a continuation of the disease, eighteen months and seven years respectively after operation, at which the removal of the tumour had been incomplete. In other cases survival periods had varied from six to twenty-three years.

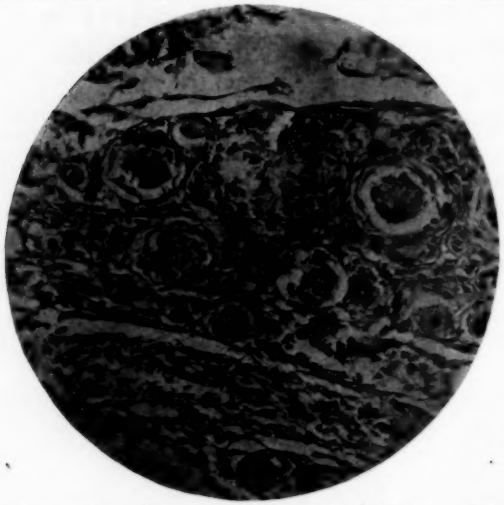

FIGURE IIA. Microscopic appearance of typical spinal meningioma. From the tumour *A* in Figure I. ×180.

A late return of symptoms in five cases has recently been reported by Davidoff, Gass and Grossman, of New York (1947). In two of their patients, women aged forty-eight and forty-one years respectively, these symptoms were due to a recurrence of the tumours (meningiomata) fourteen and twelve years after their removal. In the remaining three, women aged fifty-six, sixty-three and fifty-seven years respectively, the return of symptoms took place fifteen, three and eight years after the removal of the tumours and was due to arachnoiditis. No mention is made of how radical the removal of these tumours had been, that is, whether the associated meninges had been excised along with them.

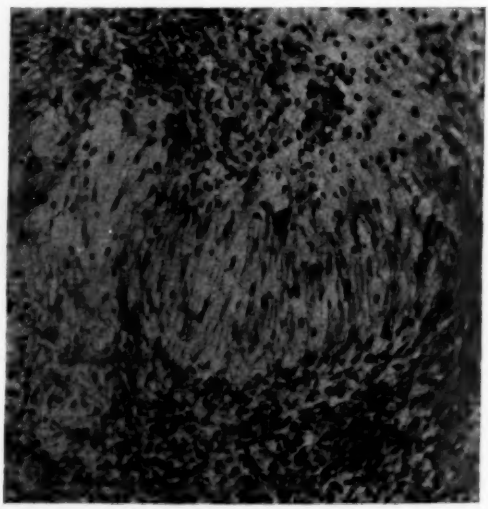

FIGURE IIB. Microscopic appearance of typical spinal neurinoma. From the tumour *C* in Figure I. ×180.

An analysis of the early cases in this unit, made from this aspect of their subsequent history, is shown in Table I.

TABLE I.

Showing Analysis of Cases of Intrathecal Extramedullary Spinal Tumour.[1]

Case.		Date of Operation.	Sex.	Age.	Occupation.	Type of Tumour.	Level.	Result.	Remarks.
Number	Name.								
1	N.M.	May 31, 1926.	F.	16	School girl.	Meningioma.	D.9	Recovery.	Well when seen October, 1929. Died of pneumonia some years later. No return of spinal symptoms.
2	W.M.	November 22, 1928.	M.	32	School teacher.	Meningioma.	D.3	Recovery.	Well and back at work until killed in 1939.
3	Mrs. E.	May 6, 1929.	F.	42	Housewife.	Meningioma.	D.2 D.3	Complete recovery. Walks normally. Lives normal life. Seen March, 1948.	States "I am wonderful", March 9, 1948.
4	H.N.	March 8, 1930.	M.	36	School teacher.	Meningioma.	D.8	Complete recovery.	Well until killed in "blitz", 1941.
5	J.R.	March 14, 1930.	M.	23	Ship's engineer.	Neurinoma.	C.1 C.2 C.3	Complete recovery. Returned to duty.	Last heard of in 1946. Very well.
6	Miss S.	March 20, 1931.	F.	59	Spinster.	Meningioma.	D.2	Complete recovery.	Well for two years. Return of pain. Re-operation, March 30, 1934: Two roots involved by arachnoiditis divided. Complete relief. Well 1948. Leading normal life.
7	W.E.A.	August 10, 1931.	M.	32	Labourer.	Neurinoma.	D.5 D.6	Recovery, but some spasticity of lower limbs persists.	Seen March, 1948. Well and gets about; gait somewhat spastic at times but no worse in recent years.
8	Mrs. C.P.	January 25, 1934.	F.	46	Housewife.	Meningioma (Anterior to cord).	D.10	Recovery.	Seen March, 1948. Very well. Walks with aid of stick.
9	F.A.	October 12, 1934.	M.	33	Fireman.	Neurinoma.	D.3	Complete recovery. Returned to duty.	Employed at fire-station. Very well. Leads normal life. Seen March, 1948.

[1] Details of these cases have been reported previously: Kennedy and Rogers (1928, 1930), Rogers (1931(*a*), (*b*)), Rogers (1935).

Of these nine patients operated upon between fourteen and twenty-two years ago, six had meningiomata, three neurinomata. Six are alive and well and three have died, one from pneumonia some years after her tumour had been removed, and the other two having been killed, one by bombing in an air raid. In the years that elapsed after the removal of the original tumour none of these nine patients has presented any signs of its recurrence. Following operation, only one has had any subsequent disturbance to an otherwise smoothly progressive and subsequently maintained state of recovery. Miss S., aged fifty-six years, two years after her tumour had been removed, had intense root pain along the course of the left ninth and tenth intercostal nerves. At a further operation I found the roots of these nerves to be invested by adhesive arachnoiditis; her pain was immediately relieved by their division and she has remained well since.

Reports by others over shorter periods of time have not shown that either recurrence of the tumour or arachnoiditis occurred as a frequent complication—Bunts (1935) 17 cases, Ingebrigtsen and Leegaard (1939) 15 cases of intrathecal extramedullary tumour.

CONCLUSION.

The favourable long-term results in the case of these spinal cord tumours removed between fourteen and twenty-two years ago is supported over a shorter period by our more recent cases. It is justifiable to conclude, therefore, that if the pressure of an intrathecal and extramedullary tumour has not damaged the cord beyond the stage at which it is capable of recovering its conduction following relief of the compression by removal of the tumour, and if early restoration of function follows operation the long-term outlook for the patient is good. Late complications in the form of recurrence of the tumour or relapse of symptoms are apparently rare. In the present series of nine cases arachnoiditis occurred as a complication in one case (meningioma). The outlook for the patient with a spinal neurinoma whose cord has not lost the power of conduction is particularly favourable. It appears from recorded cases that it is the meningioma which may recur or set up a local arachnoiditis at the site from which it has been removed, and it is advisable, therefore, to excise adequately the area of the *dura mater* to which the meningioma is attached.

SUMMARY.

Reported end results of intrathecal extramedullary spinal cord tumours have been investigated to ascertain whether recurrence of the tumours or relapse of symptoms due to other causes has taken place after a period of years.

The subsequent history of nine patients who had intrathecal extramedullary tumours (six meningiomata, three neurinomata) removed between fourteen and twenty-two years ago has been followed. One patient had root pain two years after operation due to arachnoiditis and was relieved of this following rhizotomy. None of the tumours has recurred.

The remote outlook for the patient with an intrathecal extramedullary tumour who makes a good early recovery following its removal is also good, particularly if the tumour is a neurinoma. Such recurrences or late post-operative relapses as have been reported have occurred in the case of a few meningiomata but are rare complications.

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THE TREND IN UROLOGICAL PRACTICE ABROAD.[1]

By HENRY MORTENSEN,
Melbourne.

HAVING within the past few days returned from a trip which covered many urological centres in the United States of America and in London, which included attendance at the meetings of the main urological associations and societies in America and that of the British Association of Urological Surgeons, it is felt that a brief survey of some aspects of the work seen may be of some interest and value to surgeons in Australia and New Zealand.

The subject of the correct method of treatment of prostatic enlargement still occasions considerable comment. It may be stated that a percentage of prostates are dealt with by all urologists by a transurethral method, be it by the loop or the punch. This percentage may vary from 20 to 100, depending on the leanings of the older men and the training of the more junior. The smaller prostates, those of inflammatory origin, the carcinomata requiring relief of obstruction and the fibrous bars are universally treated by this method. Argument still persists as to the necessity for resecting down to the capsule in every case—the so-called transurethral prostatectomy—but the consensus of opinion is that the more complete the removal, the less likely is the patient to develop many of the complications of this operation.

Alcock, of Iowa City, one of the pioneers of the transurethral route, is leaning more and more to a two-stage resection. In the past 1,500 cases that he has analysed, the bad risks, comprising some 450, were done in two or more stages. It was found that the mortality rate in this group was half that of the one-stage or pre-operatively better risk group.

The anuric syndrome after resection has received great notice in the past three years. Attention was first drawn to the possibility of this being due to hæmolysis caused by the forcing of water into the veins of the prostatic plexus during the progress of the operation; this would then be followed in certain cases by a blocking of the renal tubules similar to that seen in transfusion reactions. To avoid this possibility Creevy has substituted a solution of glucose as the irrigating fluid. This causes the instruments to become very sticky, and Nesbit is using a solution of glycin (1·1%). Each of these solutions produces a certain amount of clouding of the operating medium, but both of these workers are definite that the percentage of reactions, both mild and severe, has considerably diminished since their use has been instituted.

Perineal prostatectomy still has its large following in the United States of America. It is an approach to the prostate along anatomical lines and, done by experts, it is a spectacular operation to watch. It still carries, however, its percentage, albeit small, of urinary incontinence and rectal injuries, and it is a difficult operation to teach.

Suprapubic prostatectomy holds its place for the majority of prostates not dealt with by resection. Numerous modifications from the routine enucleation, with suture hæmostasis and drainage, are seen. The skin incision may be vertical or more often transverse. Some follow Millin and incise the rectus sheath transversely. Enucleation is fairly generally achieved by the intraurethral technique. Hæmostasis, if not achieved by suture, may be

[1] Accepted for publication on September 2, 1948.

secured by the use of one of the many types of inflatable bags, with or without the use of thrombin or absorbable gauze packed around it. Others pack the cavity tightly with such gauze and drain the bladder; others again were seen packing the cavity around a catheter and sewing up the bladder completely. Three cases of calculus formation with an unabsorbed portion of the gauze as a nucleus were either seen or heard discussed.

There appears to be a marked tendency in England and America towards a lessening in rigidity of the pre-operative requisites in prostatic cases and a trend towards early operation in most cases without the preliminary drainage. The general tendency in all prostate operations is to get the patient out of bed as soon as possible.

The retropubic approach to the prostate has created great interest and controversy since its presentation in America by Terence Millin at the annual meeting of the American Urological Association in 1947. Many urologists spoke on the subject at the meeting at Boston in June of this year, some, like Bacon, of Los Angeles, and Orr, of Florida, reporting 100 cases and showing excellent films in colour of the operation. Many modifications were reported, Millin himself, who was at the meeting, stating that he had heard of 24, but none was felt to be of any great consequence. This operation was seen performed by Millin in London on nine occasions and in his hands is most impressive. He is technically very skilful, makes the procedure look very simple, and rarely takes longer than twenty minutes to perform it. He still uses the transverse incision through the capsule of the prostate, whilst others (Riches, Yates Bell and Vernon) prefer a vertical incision. Ogier Ward has devised an approach which combines a vertical incision through the prostatic capsule retropubically to join up through the bladder neck with a very low vertical incision through the bladder.

The London operators using the retropubic approach are all very enthusiastic about it. They claim a surprisingly low mortality rate, a small amount of post-operative troubles, shorter bed time ("they can get up just whenever they feel like it"—Millin) and a shorter period of time in hospital. The catheter is left in by Millin for five days, by Riches two days, and since the principle of resection of the bladder neck as part of the operation has been adopted there has been no trouble with post-operative stricture of the bladder neck. The Continental urologists are equally enthusiastic. Van Gulik, of Amsterdam, told the writer of 200 cases with one death.

Discussion of the Millin operation invariably brings up the condition of *osteitis pubis*. Whilst Millin states that he has seen the syndrome quite rarely, many cases were reported following the operation at the various meetings attended, and the impression was gained that the incidence was relatively high. That the condition is not purely a complication of retropubic prostatectomy is well known, and although only recently seen or recognized to any great degree, it was reported after such operations as total or partial cystectomies, suprapubic prostatectomy, pelvic ureterolithotomy and even a simple fall on the pubes. One of the most instructive films seen was that of a patient suffering from this distressing complication of pelvic surgery attempting to get out of bed and walk. No one seeing the obviously painful and futile attempts of this man to move would ever forget the possibility of its occurrence. The pathology was obviously little understood and treatment comparatively ineffectual. It was reported that irradiation with X rays offered perhaps the greatest possibility of amelioration of symptoms.

At the meeting of the Association of Genito-Urinary Surgeons at Skytop, Pennsylvania, an interesting paper was read by Jewett, of Baltimore, on the results of radical surgery in the treatment of carcinoma of the prostate.

He had analysed the cases in which operation had been performed at the Johns Hopkins Hospital, Baltimore, prior to May, 1943, of which there were 132 in all. Of 54 with extracapsular extension revealed either by macroscopic or microscopic observations, three are living ten years or longer and seven for a period of six to nine years. Of 78 with no evidence of extracapsular extension, 24 have had recurrence. Of the remainder, 40 lived or are living five years or longer without evidence of cancer, and 12 a period of 10 to 27 years.

Millin told the writer that he had performed a radical prostatectomy by the retropubic route in several cases with satisfactory results and ease of operation.

Apart from radical removal, the routine treatment of prostatic carcinoma appears to be resection of tissue causing obstruction when this symptom is not relieved by œstrogenic therapy, the withholding of such therapy until symptoms become pressing again, then the use of stilbœstrol in doses that range from one milligramme per day to one hundred milligrammes per day, followed, if results are unsatisfactory, by orchidectomy.

Malignant tumours of the bladder were discussed at great length. Reynolds, Schulte and Hammer, of San Francisco, elaborated on their method of dealing with serious tumours by the use of the loop resectoscope to remove transurethrally the mass of the tumour down to muscle fibres and even to fat with coagulation of the base. They claim for this procedure results comparable with or better than those obtained from other more radical methods.

Complete removal of the lesion by total cystectomy is a popular method of approach in both America and England.

Diversion of the urinary stream is achieved by either cutaneous ureterostomy or transplantation into the sigmoid colon.

In an attempt to make the post-operative handling of the ureteric stump in the former operation more satisfactory, Fish, of New York, is attempting to bring the ureter through a tunnel made through a finger of skin constructed at a previous plastic operation. This permits the comfortable wearing and fixation of a drainage appliance. Other operators use bags of various types or cups which are sealed to the skin, in an attempt to produce dryness, and these need to be changed weekly.

In the main, the methods of ureteric transplants are tending to become more simplified. The extraperitoneal approach popularized by Priestley, of Rochester, Minnesota, has almost completely been superseded by the original intraperitoneal method, although the former operation was seen performed by Riches, of London, who claimed for it very satisfactory results. The method generally consists of a short tunnelling of the muscular coat of the bowel, a comparatively large opening into the bowel, and the ureter drawn down as far as possible into it by means of a long needle threaded through the distal end of the ureter, brought out through the bowel and fixed.

In this regard greatest interest centred around the attempts of Nesbit, of Ann Arbor, Michigan, to achieve direct anastomosis of the ureter to the bowel. An incision is made longitudinally through the distal end of the dissected ureter and at a suitable site an incision to correspond to the length of the opening of the ureter is made into the colon. Direct anastomosis of the wall of the ureter to the wall of the bowel is then made. Experimentally the results on dogs have been very encouraging, all the dogs living, a unique occurrence in animal transplants. Twelve operations have been carried out in humans, the youngest being a child of eight days, with no deaths, a temperature rise in only one case, and with post-operative pyelograms showing in every case but one either no upper tract dilatation or dilatation to a

minimal degree. Further results of this procedure will be awaited with great interest. The method of Flocks, of Iowa City, in which the ureter is laid in its colonic bed, sutured to the bowel, but not severed in its continuity till some days later, whilst more complicated, has been followed in his hands with excellent results in a much larger series of cases.

In an attempt to place the operation of total cystectomy for carcinoma on a basis similar to that of cancer surgery in other parts of the body, an endeavour is being made to obtain a dissection of the adjacent lymph gland fields by stripping the great vessels of the pelvis of their glands and areolar tissue. This is made a routine portion of the procedure of total cystectomy by Higgins, of Cleveland, who quotes cases of patients living and well for periods of five years or more with proven gland involvement at operation, and of two patients who had died from other causes in whom post-mortem examination five years after operation revealed no trace of malignancy. Marshall and Whitmore, of New York, were seen adding to a total cystectomy the removal of the rectum in an attempt to facilitate this cleaning out of the pelvis in a bladder tumour of great histological malignancy.

In other centres the enthusiasm for the curative possibilities of cystectomy was not so great. It was stated at the Mayo Clinic that any dilatation of the upper urinary tract which could be reasonably ascribed to a bladder malignancy rendered the case incurable. On the other hand, one saw Higgins bring a dilated ureter to the skin, as a preliminary method of drainage of the dilated kidney pelvis and ureter, with subsequent anastomosis into the bowel.

From an experimental point of view the prize essay read by Seymour Rubin, of Baltimore, entitled "The Formation of an Artificial Bladder with Perfect Continence", was outstanding. In dogs an artificial sigmoid bladder was constructed experimentally into which the ureters were transplanted, total cystectomy was performed and the new bladder anastomosed to the proximal urethra. The dogs were able to void with perfect control.

In the surgery of renal tumours great interest was evinced in the procedure of transthoracic nephrectomy, first reported by the writer in 1947, for use in tumours whose removal would provide gross difficulty by the previously accepted methods of approach. Chute, of Boston, reported three difficult tumours removed by this method and showed a film of the procedure, and subsequently V. J. O'Conor, of Chicago, used this approach in a case of tuberculous kidney on the right side, with a very marked kyphosis due to an earlier healed spinal lesion. The patient in this case had been refused operation for over two years and O'Conor was delighted with the ease of this approach.

Mainly owing to the writings of Lloyd Lewis, now of Washington, D.C., whose experience in army hospitals and since the end of the war in veterans' administration institutions has been vast, there is in America a definite swing back to the radical dissection of glands in association with orchidectomy in the treatment of tumours of the testicle.

Lewis states that simple orchidectomy should be reserved for cases of benign interstitial cell tumours and that for teratomata radical orchidectomy is indicated without radiation. For all other tumours radical orchidectomy is indicated, followed by post-operative therapy, the dosage varying with the pathological nature of the tumour from 1,000*r* in seminoma to 5,000*r* in trophoblastic tumours where inoperable nodes occur. It will be interesting to hear of his five-year results. Hinman, one of the pioneers of the radical operation, has now returned to it in selected cases.

In the early diagnosis of tumours of the urinary tract the recognition of individual tumour cells in the urine by means of the Papanicolaou

technique has received considerable attention. In bladder tumours this may be of definite value, but in the diagnosis of renal tumours false positives are still producing errors in diagnosis and in some cases unnecessary nephrectomy. The cells of prostatic carcinoma in the prostatic secretion are more readily identified, and this technique may provide a useful aid to the diagnosis in doubtful cases.

For severe cases of interstitial cystitis, the so-called Hunner ulcer which fails to respond to the simpler treatments of hydrostatic dilatation, fulguration by diathermy or treatment with silver nitrate, Nesbit has had the operation of chordotomy performed with excellent results.

The use of streptomycin in the treatment of tuberculosis was discussed at many centres. It would appear that the greatest value of this antibiotic would lie in the comparative control of bilateral cases. Alleviation of the distressing symptom of painful frequency may be expected. Its use after nephrectomy in unilateral cases will lead to rapid healing of bladder lesions with relief of symptoms and disappearance of the organism from the urine, but will have no effect on the interstitial cystitis which often is a cause of frequency after the cure of the disease. It must be remembered that in its use fibrosis takes place so rapidly around the lesion that obstructive lesions may be produced in the urinary tract. Cases where as a result of complete obstruction to the normal ureteric orifice in bladder healing retransplantation of the ureter into another site in the bladder had to be undertaken were reported by O'Conor. The dosage used by various observers differs, the standard being one gramme per day. Nesbit, however, reports that in nineteen patients on a dose of two grammes per day there has been no recurrence of tubercle bacilli in the urine over a period of six months, whilst in three with one gramme there was recurrence in all. Three cases of ureteric stricture occurred in his series. Massey, of Pasadena, claims results with a dosage as low as 0·25 gramme per day.

The use of chaulmoogra oil in association with streptomycin following the lines of Slotkin, of Buffalo, leads to a lessening of dose and an enhanced result.

The use of soap containing G.11, first reported by Lockwood in *Surgery*, October, 1947, bids fair to revolutionize the pre-operative preparation of the surgeon's hands. Thirbly, at Ann Arbor, working under Nesbit, is conducting a thorough investigation and is satisfied that the growth of bacterial flora on the hands after its use is considerably diminished compared with the result obtained with other methods. Preparation with this soap requires a lathering of the hands and forearms for one minute without scrubbing. The lather is then washed off, the nails are cleaned with a sterile orange stick, and the process is repeated for another minute, after which time the required degree of surgical asepsis has been reached. No irritative effects on the hands have been noticed.

Case Reports.

CARCINOMA OF THE COLON WITH SPONTANEOUS FÆCAL FISTULA.[1]

By Howard H. Eddey,
Melbourne.

Clinical History and Operative Details.

W.G., aged sixty-two years, was admitted to the Royal Melbourne Hospital on July 2, 1946, with a history of having had a left inguinal hernia for the previous seven years. This hernia was readily reducible at first, but became irreducible three months prior to his admission. On July 1, 1946, the hernia increased in size, became red but not painful, and during the night broke down and discharged copious quantities of fæces. The appetite and digestion had always been good, but there had been a loss of two stone in weight during the preceding two months. The bowels were slightly constipated until the day before admission, when they became loose.

Examination showed a healthy looking elderly man. His temperature was 99·4° F., his pulse rate was 96 and his respiratory rate 24 in the minute. The tongue was slightly coated and the breathing abdominal. There was a large irreducible left inguinal hernia filling the left side of the scrotum. A fistulous opening, half an inch

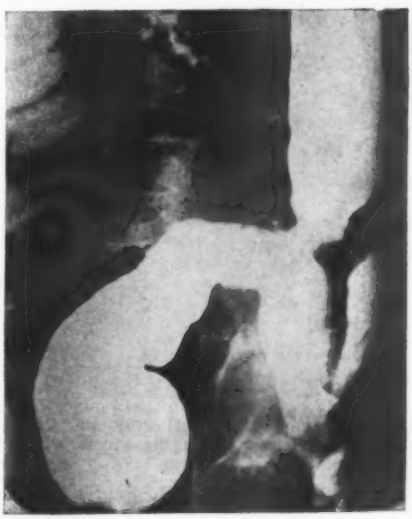

Figure I. Skiagram of the final state.

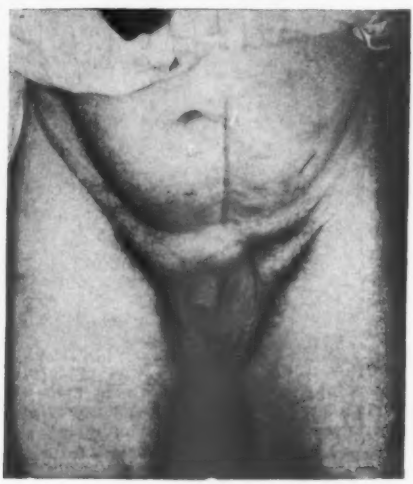

Figure II. Showing the end result.

in diameter, was present on the summit of the hernia; out of this opening fæces were exuding. The skin over the hernial sac was reddened in places.

The first operation was performed on July 5, 1946. Anæsthesia was induced by ethyl chloride and "open" ether. An incision was made over the hernial sac. When the sac was opened it was found to contain large quantities of fluid fæces and the loop of sigmoid colon, and its walls were gangrenous. The sigmoid was separated from the sac and lifted outwards. At the apex of the loop was an annular carcinoma which had perforated at its mesenteric side, and there was a second perforation, also

[1] Accepted for publication on May 17, 1948.

discharging fæces, near the neck of the sac. The fæcal matter was cleared out of the sac, the apex of which was found to have sloughed; the scrotal septum was also gangrenous, so that both halves of the scrotum communicated. A wide rubber drain tube was introduced into the sigmoid colon through the perforation near the neck of the sac and the loop was exteriorized. Counter incisions were made to drain the scrotum and the hernial sac.

During the post-operative course there was copious drainage of fæces from the perforated apex of the bowel, but very little by the drain tube; large sloughs were present in the various incisions and these were dressed frequently.

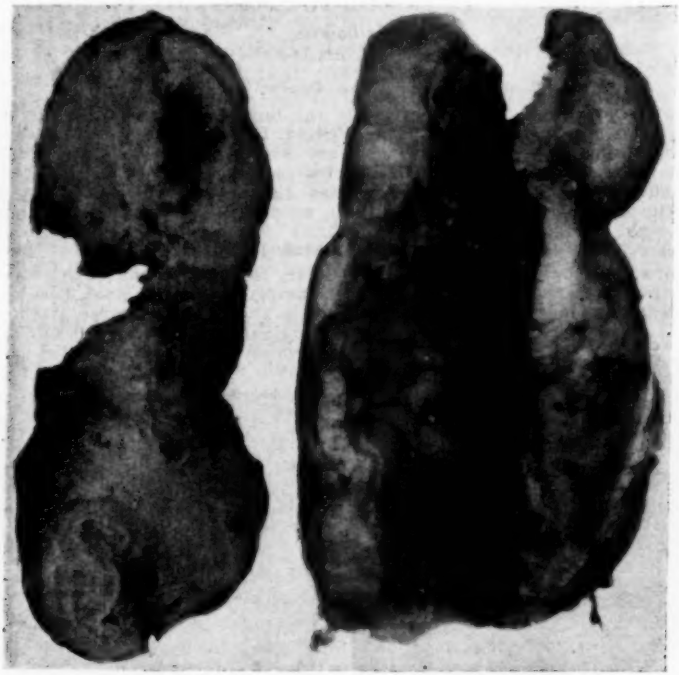

FIGURE III. The macroscopic specimen.

The second operation was performed on July 12, 1946. "Pentothal" anæsthesia was employed. The exteriorized loop of bowel was removed by diathermy and hæmostasis was secured. The loop measured eight inches in length, with the carcinomatous ulcer present exactly in the middle.

Post-operatively the wound drained freely, but became healthy looking after the discharge of sloughs.

The third operation was performed on August 14, 1946. Anæsthesia was induced by ethyl chloride and "open" ether. Through a left paramedian incision the two limbs of the colostomy were identified; the mesentery of this portion of the colon contained a mass two inches by one inch in size, which appeared inflammatory in nature. There was no evidence of metastases in the abdomen. The proximal limb of the colostomy was divided between clamps about three inches from the colostomy opening. The distal segment of this limb was oversewn and an end-to-side anastomosis was made between the proximal segment and the distal limb of the colostomy. The final state is shown in the X-ray photograph (Figure I). A tube was passed down to the site of anastomosis through a stab wound in the outer part of the iliac fossa and the paramedian incision was closed. Despite attempts to clear the rectum and distal loop of the sigmoid pre-operatively, the distal loop contained considerable quantities of fæces at operation.

Continuous intravenous administration of 5% glucose and saline solution and a course of sulphamerazine were given post-operatively and the patient was placed on a non-residual diet. In the first week the original colostomy opening drained fæces copiously, probably because of the loaded rectum and distal sigmoid, no attempt being made to empty them by enemata because of danger to the suture line. After ten days enemata were given and following these the fæcal discharge at the colostomy rapidly diminished and the bowels acted normally. The paramedian incision healed by first intention.

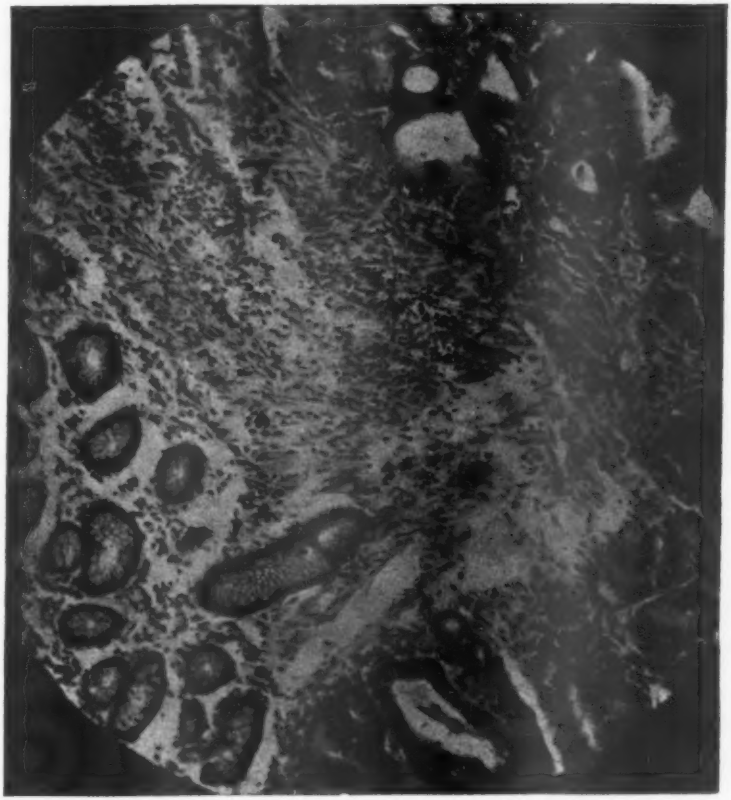

FIGURE IV. Microscopic appearance of specimen.

By August 30, 1946, there was only an extremely small fæcal discharge from the distal limb of the original colostomy, which by this time opened by a small aperture at the bottom of a puckered scar in the original hernial incision. The final state is illustrated in Figure II.

The patient was discharged from hospital on September 6, 1946, and by December 20, 1946, he was very well, had gained three stone in weight and was working. His bowels acted normally one to two times a day, and only a small amount of fæces was discharged onto the surface, the discharge ceasing completely for days at a time. By June 20, 1947, the discharge from the original colostomy had ceased for some months; there was no evidence of recurrence of the hernia or of metastases in the abdomen or elsewhere.

Investigations.

The following investigations were carried out.

1. A barium enema was given on August 27, 1946. Dr. B. G. Wood reported as follows:

> The large bowel filled readily to the caecum; the descending colon is anastomosed to the side of the pelvic colon with a stoma of about three-quarters

of an inch in width. The loop of the pelvic colon is open proximally at the colostomy, where there is also a blind pouch running upwards and slightly laterally from the colostomy. At the blind end of the pouch there is a fine branching sinus. There is also an old central dislocation of the head of the left femur with considerable hypertrophic bone formation and gross arthritic changes.

2. Pus from the slough in the lower margin of the hernial incision was examined by culture methods on July 19, 1946; a profuse growth of pneumococci, *Bacillus coli* and *Bacillus pyocyaneus* was obtained.

3. A pathological report was received on July 12, 1946, from Dr. I. C. Heinz, as follows:

> Portion of greatly œdematous bowel with thickened wall and containing an abscess cavity. Three small polypi are growing from the mucosa; one is in the centre of an ulcer with heaped-up round edges which encircles the bowel over a distance of one and a half inches. Microscopically, adenocarcinoma. The two separate polypi are simple in nature.

Photographs of the specimen are reproduced (Figures III and IV). The blue pointer is placed in the hole where the abscess cavity has ruptured at the mesenteric border of the bowel, thus allowing discharge of fæces into the hernial sac. The two separate polypi are seen, one just above the colourless rod and the other three-quarters of an inch below it. Also reproduced is a cross-section through the two limbs of the sigmoid loop and its mesentery, showing the gross œdema of the loop and the spread of growth into the mesentery. The photomicrograph shows the edge of the carcinoma demonstrating the gradual change from normal bowel mucosa into adenocarcinoma. The polypus in the centre of the carcinomatous ulcer is filled with malignant acini.

Comment.

When the sac of a left inguinal hernia contains portion of the sigmoid colon it is not particularly uncommon to find that there is a carcinoma present in this portion of the sigmoid. However, it must be most uncommon for a patient to seek medical advice after the carcinoma has perforated and a fæcal fistula has formed. No report of a similar case could be found in the literature.

It is surprising that the fluid fæces did not escape through the neck of the sac into the peritoneal cavity before the skin over the hernial sac broke down to form a fæcal fistula. Complete adherence of the peritoneum to the bowel at the neck of the sac must have prevented much leakage.

Although the skin incisions have now healed and there is no fæcal discharge, it is felt that, should mucus collect in the oversewn distal segment of the proximal limb of the sigmoid, the mucus will discharge through the thin skin scar and not through the blind stump of the bowel, and therefore no further surgery on this blind pouch of bowel is contemplated.

The occurrence of three discrete polypi, two of which are innocent and one of which is filled with malignant acini and lies in the centre of the carcinomatous ulcer, illustrates the frequent association of discrete polypi and carcinoma of the colon.

ADVANCED CARDIOSPASM: THREE CASES IN WHICH OPERATION BY THE THORACIC ROUTE WAS PERFORMED.[1]

By Douglas Robb and Rowan Nicks,
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Inasmuch as the name of one of us has appeared twice already in this journal in case reports of advanced cardiospasm, and because we have found a fresh approach and simpler methods to offer considerable advantages, we feel that some details of three further cases may be recorded.

The first patient, Mrs. S., aged forty-eight years, had had dysphagia for fifteen years. The condition had been accurately diagnosed but minor treatment with bougies, œsophagoscopy *et cetera* had given no relief whatever. She was finally referred for treatment to the Green Lane Hospital Thoracic Unit by Mr. Eric Macdonald and Dr. Mollie Wagstaff, of Dargaville. There was the usual gross dilatation and elongation of the œsophagus. Cardioplasty was advised.

The second patient, Mr. W., aged seventy-five years, was referred by Dr. K. H. Holdgate. He had had severe dysphagia for forty years. He was emaciated, and his heart was fibrillating. The œsophagus was really gross in size, appearing to fill the right hemithorax after barium was taken. Considerable time and trouble were spent in trying to improve the patient's nutritional state, and also his cardiac condition, without much success. Eventually operation was undertaken.

The third patient, Mrs. R., aged thirty-nine years, had a ten-year history of dysphagia, increasing in severity and unrelieved by periodic œsophagoscopy and dilatation. Radiologically the gullet was not so grossly enlarged as in Case I and had not yet developed a sigmoid loop in its lower part. It was wide throughout, and filled up to the clavicles with barium, though at that level discharge was taking place into the stomach. Operation was advised because of the early age, the long and progressive history, and the failure to relieve by minor measures.

Pre-operative preparation in all cases was similar. Nutrition was studied; ascorbic acid, sulphadiazine and penicillin were given, and also breathing exercises. For local cleansing and emptying of the dilated gullet, the extreme head-down position on the rocking stretcher, used and described for bronchiectasis by Dr. Chisholm McDowell, head of the Green Lane Chest Unit (medical side), was employed. In the first case irrigation of the gullet through a swallowed tube was used in that position, but in the others the posture alone was sufficient to empty the gullet.

For operation the thoracic route was chosen in all cases. "Intratracheal cyclopropane" and oxygen were used and fluid and blood were given intravenously as required. The ninth rib on the left side and an inch at the angle of the eighth were resected, and a good view of the lower part of the œsophagus was obtained by lightly retracting the lung. Reinflation of the lung was carried out by the anæsthetists (Dr. Anson and Dr. Innes) at twenty-minute intervals throughout the operations, which lasted 130 minutes from skin incision to skin suture in the second case, a little longer in the others. The œsophagus was mobilized and a tape passed around it.

In all cases there was a narrow tapered waist a little more than one centimetre wide above the œsophageal opening in the diaphragm before the widening to form the stomach. The lower part of the œsophagus was massive and muscular in each case, but at the waist there was a notable absence of any muscular thickening or fibrous stricture in any coat. In the first case but not the others there was a small mucosal web at the junction.

The œsophageal opening in the diaphragm was enlarged forwards, several diaphragmatic vessels requiring ligature and division. The muscular crus on the under surface was divided and the peritoneum opened.

A longitudinal incision, about seven centimetres long, was made into the lumina of gullet and stomach, centred at the waist. Considerable venous oozing took place from the muscular and submucous coats, ligatures being required. This incision was sewn up transversely in three layers, beginning with mucosa, by interrupted cotton sutures, knots on the lumen. The muscular layer was substantial. The third layer united the

[1] Accepted for publication on February 26, 1948.

outer muscular coat of the œsophagus to the peritoneum of the stomach. Care was taken at all stages to avoid any unnecessary trauma to the delicate tissues of the œsophageal wall. A good lumen could be palpated through the walls on the completion of the suturing.

The diaphragm was partly resutured and the remaining edges were stitched to the stomach wall. Penicillin and sulphathiazole powder was applied to the region of the suturing and the œsophageal bed. An intercostal tube to an underwater seal was inserted and the wound completely closed.

Post-operatively no troubles were encountered. Even in the second case the procedure was well tolerated. Reexpansion and full inflation of the lung were not difficult. Fluids by mouth were given cautiously, reliance being placed on intravenous administration of glucose for several days. Natural drainage of the gullet through the new opening was good, and there was no need for inlying tubes in gullet and stomach as formerly used. All patients were got out of bed early and were ready to go home in fourteen days from the operation. All were enormously relieved by the improvement in their swallowing, which was effortless and painless. Their general condition and state of nutrition improved rapidly.

Summary.

Three further patients with advanced cardiospasm have been operated on, making five in Auckland in the past seventeen years, the only ones known to the writers.

Though the abdominal route has proved quite adequate, the thoracic is now favoured, on account of the excellent access and the greater facility and accuracy possible in suturing the new opening.

Pre-operative preparation of the gullet by lavage and emptying is facilitated by the use of the rocking stretcher.

Post-operative management has been simplified by abandoning the use of inlying tubes in the gullet. Ordinary post-thoracotomy management must be carefully applied.

Acknowledgement.

One of the writers (R.N.) acknowledges the guidance and help of Mr. N. R. Barrett, London, and Dr. Richard Sweet, Boston, in this matter.

Post-Scriptum.

Since this memorandum was prepared a fourth patient with cardiospasm and one with peptic ulcer at the junction of the gullet and stomach with a sizable portion of stomach lying in the chest above the opening in the diaphragm have been operated on by this thoracic route. A second case of similar peptic ulcer also has been dealt with in association with Mr. F. P. Furkert, F.R.C.S. In all cases access proved easy and confirmed the writers' present preference for the thoracic route.

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PYLORIC STENOSIS WITH AZOTÆMIA.[1]

By R. M. Dunn,
Sydney.

In January, 1923, Haden and Orr, in the *Bulletin of the Johns Hopkins Hospital*, described three cases of acute intoxication following gastro-enterostomy. They ascribed the symptoms as being due to absorption of a toxin from a closed upper intestinal loop. Later in the same year Brown, Hartman, Eusterman and Rowntree[(1)] described a series of similar cases.

In 1928 French writers Blum and Rathery again noted the uræmic changes associated with hypochloræmia (*urémie par manque de sel*). The only reference found in Australian literature is a comprehensive article by Falconer and Lyall, in The Australian and New Zealand Journal of Surgery in 1938.[(2)] They give a good account of symptomatology and point out the advantages of jejunostomy.

Apart, then, from the few cases reported in the surgical literature, very little progress has been made in finding a satisfactory explanation for the syndrome in the past twenty-five years.

The four cases reported here were all seen in the past twelve months at Concord Military Hospital. They were all known cases of duodenal ulceration. Azotæmia followed pyloric stenosis due to ulcer in three of the cases, and in the other case followed vagotomy and posterior gastro-enterostomy (Case III).

All the patients had also been under medical observation and treatment for some weeks at least before their profound biochemical upset forced some sort of surgical procedure.

The lesson learnt was that any appreciable degree of pyloric stenosis calls for early surgical relief before vomiting *et cetera* become intractable. Alkalinization, antispasmodics, gastric lavage *et cetera* have but little effect on organic obstruction, and it is but rarely that this treatment can release the accompanying pylorospasm.

If it is not known that the patient has an ulcer diagnostic difficulties may be considerable and the cause of the azotæmia may be concealed or considered as renal in origin. The usual sequence of events is a healing prepyloric or duodenal ulcer which has been present for months or years (sometimes with remarkably few symptoms). Signs of obstruction then appear (in one case in this series following suture of ruptured ulcer) with increasing vomiting. After some period of slow starvation obstruction becomes absolute from inflammatory œdema and swelling. The hypoproteinæmia serves to increase the pyloric œdema.

Case Histories.

Case I.

A.D., a male, aged twenty-four years, gave a history of dyspepsia which began in New Guinea in 1943. A ruptured duodenal ulcer was oversewn at 113 Commonwealth Military Hospital in May, 1945. Soon after recovery from this he began to get attacks of vomiting, which had recently become worse, and on his admission to hospital were almost continuous. No bleeding occurred. The patient had lost approximately one and a half stone in weight.

On April 9, 1947, he was admitted to 113 Commonwealth Military Hospital. He was very drowsy, with hiccup *et cetera*. His systolic blood pressure was 120 and his diastolic pressure was 70 millimetres of mercury. Carpopedal and facial spasm was present (tetany).

On April 21, 1947, a barium meal revealed marked dilatation of the stomach, which contained a large fasting residue (about ten ounces).

On April 29, 1947, the blood urea content was 76 milligrammes *per centum*; the blood chloride content was 382 milligrammes *per centum*; the plasma bicarbonate content was 85 volumes *per centum*; ten ounces of urine were passed in twenty-four hours. The urine was alkaline; it contained no chlorides, but a trace of albumin and an occasional cast. During this period antispasmodics, gastric lavage *et cetera* produced no improvement.

On May 26, 1947, the patient was examined by a surgeon and intravenous therapy with saline solution *et cetera* was commenced.

[1] Accepted for publication on June 10, 1948.

On May 29, 1947, posterior gastro-enterostomy was performed. Convalescence was normal and complete relief of symptoms occurred within five days. His subsequent history has been uneventful.

Comment.—In this case pyloric stenosis became established after suture of a ruptured ulcer two years previously. This is probably a not uncommon event. His blood chemistry rapidly returned to normal with suitable therapy and subsequent gastro-enterostomy was uneventful.

This patient ran the gamut of hypochloræmia, alkalosis and tetany and finally uræmia. He was treated medically for at least one month before operation, without success. He sustained no permanent renal damage.

Case II.

F.F.L., a male patient, aged thirty-four years, had epigastric pain and vomiting, which began in attacks in 1942. He had vomited blood on several occasions and was in hospital several times. This state of affairs continued until December, 1946, when he was admitted to hospital with a history of incessant vomiting for the previous five days. He had lost two stone in weight.

On December 20, 1946, on examination, he was drowsy, his tongue was dry and furred. His systolic blood pressure was 110 and his diastolic pressure was 80 millimetres of mercury. His pulse rate was 120 in the minute. He had some abdominal cramps and his hands showed intermittent carpopedal spasm. He passed ten ounces of urine in the twenty-four hours; it contained no chlorides, but a trace of albumin, and no casts. The blood urea content was 200 milligrammes *per centum*. Intravenous therapy was commenced with normal saline solution and glucose.

On December 24, 1946, the urinary output was satisfactory. The patient was still drowsy.

On December 27, 1946, the blood urea content was 112 milligrammes *per centum*. The patient's condition was much improved.

On January 3, 1947, the operation of posterior gastro-enterostomy was performed; it was complicated by right basal collapse. In the subsequent two months the patient rapidly regained his weight and felt very well.

Comment.—The patient had a good early result following gastro-enterostomy. It is interesting to note that his acid curve rose sharply when anastomosis was established and gastritis had subsided. This patient also had had medical treatment for some weeks without effect.

His subsequent history is interesting if irrelevant. Six months after operation he had a further severe hæmatemesis, for which partial gastrectomy was performed. Two months later his pain recurred and a stomal ulcer is now demonstrable. It is probable that he will require a vagotomy.

Case III.

S.B.T., a male, aged twenty-six years, complained of epigastric pain, which began in 1942. Two months later his appendix was removed with relief of symptoms. In 1944 the pain recurred. Six months before he was seen pain began again and gradually became worse. Vomiting commenced and was present after almost every meal. He had lost one stone in weight in the previous six months.

On February 19, 1948, a barium meal revealed marked deformity of the duodenal bulb, with considerable delay in emptying. There was a residue after thirty-six hours. The usual medical treatment was commenced, but vomiting persisted in spite of this and the patient was seen by a surgeon on March 20, 1948.

On examination considerable dehydration was present. Oliguria had been increasing over the past week. The urinary chloride content was less than one gramme per litre. The serum chloride content was 540 milligrammes *per centum*. Intravenous therapy was commenced with saline solution and glucose.

On March 23, 1948, the operation of subdiaphragmatic vagotomy was performed with posterior gastro-enterostomy.

On March 25, 1948, there was a gastric residue of 300 ounces.

On April 1, 1948, the serum chloride content was 510 milligrammes *per centum*. The blood urea content was 70 milligrammes *per centum*. The patient had well-marked gastric dilatation following vagotomy. The stoma was completely obstructed. Clay-coloured stools were passed. Gastric aspiration was performed for two weeks following gastro-enterostomy without effect. The gastric residue during this time was never less than 100 ounces per day.

On April 3, 1948, the operation of jejunostomy was performed under local anæsthesia and cyclopropane. Approximately 3,000 Calories per day, together with adequate

protein and carbohydrate, bile salts and pancreatic extract, were fed through the jejunostomy tube. Improvement followed this, but upper intestinal obstruction continued (presumably due to gross gastric dilatation pulling up and kinking the stoma behind the transverse colon). Anterior gastro-enterostomy was performed a fortnight later, with complete relief of symptoms.

Comment.—In view of the experience in Case II we were persuaded to perform a posterior gastro-enterostomy and subdiaphragmatic vagotomy in this case. This was a mistake, as at no time did the stoma function, owing apparently to marked gastric atony and dilatation.

It was only at this stage that the patient became uræmic. Jejunostomy provided temporary relief and was a very useful measure. However, the patient did not gain weight and the added pancreatic and biliary secretions did not seem particularly effective.

In future we would not perform gastro-enterostomy (particularly the posterior anastomosis) and vagotomy in the presence of gross gastric distension and hypoproteinæmia.

Case IV.

J.C.M., a male, aged twenty-seven years, suffered from a duodenal ulcer diagnosed two years previously. He had a recurrence of symptoms several months previously, particularly vomiting, which had been frequent and persistent. On April 4, 1947, he was readmitted to hospital. Vomiting persisted. He had lost one and a half stone in weight. Medical treatment was commenced with alkalis *et cetera.*

On April 14, 1947, a barium meal showed that the stomach was grossly distended with residue and the appearances suggested pyloric stenosis due to old ulcer.

On April 15, 1947, gastric suction was commenced. On April 22, 1947, the residue was 78 ounces. The patient was seen by a surgeon. Intravenous therapy was commenced with blood plasma and saline solution.

On April 23, 1947, the gastric residue was 72 ounces. The urinary output was very poor. The blood urea content was 170 milligrammes *per centum.*

On April 24, 1947, posterior gastro-enterostomy was performed under local anæsthesia. A liver biopsy was also taken. A tube was put through the anastomosis and the patient was fed with "Varco" diet and "Triamino" *et cetera* (3,000 Calories, 150 grammes of protein per day).

On April 25, 1947, the patient had signs of marked peripheral failure with cold bluish extremities *et cetera.* No urine had been excreted since operation.

On April 28, 1947, there was no improvement in the patient's condition. The gastric residue was 200 ounces. The urinary output was 16 ounces. The blood urea content was 229 milligrammes *per centum.*

On April 30, 1947, the patient died with congestion at the bases of both lungs; he was deeply cyanosed and almost anuric.

Post-mortem examination was carried out at 1030 hours on May 1, 1947. The body was that of a well-developed young man. *Rigor mortis* and post-mortem staining were present.

In the heart the right auricle and right ventricle were dilated. The left ventricle was normal. The valves were normal. The coronary arteries were patent. The muscle walls showed no abnormality.

Both lungs showed extensive bronchopneumonia, most marked in the lower lobes. In addition there was much hæmorrhage and œdema.

In the abdomen the stomach was distended. A recent posterior gastro-enterostomy had been carried out. There was slight extravasation of blood at the margin of the stoma, but no leak through had occurred.

In the first part of the duodenum, at its junction with the pylorus, there was a moderate degree of stenosis of the wall. There was no obvious ulceration. The small intestine was dilated and congested, and contained a few fibrinous flakes on the surface.

The liver was paler than normal. A small subcapsular hæmatoma, one and a half inches in diameter, was present in the under surface of the liver (traumatic in origin).

The gall-bladder and pancreas were normal. The spleen was slightly enlarged and congested. The adrenals and the kidneys were normal.

Examination of a liver section showed necrosis in the central portion of hepatic lobules. The condition was focal necrosis (zonal). Examination of a kidney section showed no gross abnormality.

A diagnosis of bronchopneumonia and duodenal stenosis was made.

Comment.—This patient never seemed to get into adequate condition for surgery and the mistake was made of doing a gastro-enterostomy instead of a preliminary jejunostomy. It should be noted, however, that jejunostomy was not an outstanding success in the previous case (III), the patient failing to gain weight *et cetera.*

He received medical treatment for far too long before he was submitted to surgery. Operation did not seem to relieve his obstruction, and gastric dilatation and atony, oliguria and uræmia persisted. He received adequate pre-operative intravenous therapy.

He died of advanced peripheral failure, with congestion at both lung bases, possibly due to too much intravenous fluid. At post-mortem examination the lack of gross microscopic changes in the kidneys was remarkable. Well-marked liver changes were, however, present and were probably responsible for his continued uræmic state.

Treatment.

It was amply shown in this series of cases that persistence in medical treatment serves but to increase the difficulties. Fluid and electrolyte balance must be reestablished with appropriate intravenous therapy. It is obvious that the adverse nitrogen balance of weeks cannot be reestablished in days. Nevertheless, pooled serum, intravenously administered protein hydrolysates and whole blood can be usefully employed. Routine posterior gastro-enterostomy is the operation of choice in the milder cases. Jejunostomy can be effectively used in the severe cases and is a useful preliminary to the above.

Vagotomy has no place in the treatment of this acute condition and its dangers have already been pointed out.

Discussion.

Uræmic intoxication can, of course, occur after any prolonged or severe fluid loss. The better known forms occur after severe burns, hæmorrhage *et cetera.* As in the cases reported above, it can also follow pyloric stenosis, gastro-enterostomy, stomal difficulties, acute gastric and duodenal fistulæ, acute dysenteries *et cetera.*

The clinical picture resembles uræmia due to any other condition. Thirst, headache and irritability are early symptoms, accompanied by dry tongue and skin. Blood pressure is usually low, owing to reduction of circulating fluid. This differentiates the renal conditions where the blood pressure may be raised.

Later the patient becomes drowsy and uncooperative, and signs of tetany may be present. The blood chloride content is reduced and chloride disappears from the urine. The blood alkali reserve rises, as does also the blood urea content. The urine is scanty and usually alkaline in reaction. It may contain albumin in small amounts, also red cells and an occasional cast. The renal changes are only temporary.

When peripheral vascular failure occurs it is terminal and irreversible.

The basic factors involved in the syndrome appear to be quite unknown, and various intestinal toxins (duodenal toxæmia of Haden and Orr), toxins due to protein breakdown, hypochloræmia, have all been cited as possible causative agents. It is well known that impairment of renal function occurs whenever a severe degree of shock develops. There seems little justification for the term hypochloræmic uræmia, and it seems that depletion of blood chlorides is but an accompaniment of the condition. Electrolytes pass readily by the diffusion in either direction through a semi-permeable membrane; hence it is difficult to see by what mechanism the hypochloræmia could effect the formation of urine.

It is also well known that tetany can follow an established alkalosis, the rise in blood pH apparently causing a diminution in the number of circulating calcium ions. Alkalosis is a necessary concomitant of chloride depletion.

The dynamics of the renal disturbance seem clear enough. The increased concentration of the blood and the decrease in circulating volume with decrease in blood pressure both reduce the effective renal filtration pressure, because the rate of filtration varies directly with the capillary blood pressure and the osmotic pressure of the plasma proteins opposes filtration.

It would appear that anoxæmia was responsible for the parenchymatous renal changes. In the death described (Case IV) there were no significant microscopic renal changes. Case IV illustrated marked liver damage, circulatory failure and terminal pulmonary congestion (possibly due to over-enthusiastic intravenous therapy).

Moon[3] points out that parenchymatous liver changes do occur with marked degrees of shock, and possibly similar factors are at work in these cases. Attempts to demonstrate a toxic factor in the blood have almost universally failed. Moon states: "Capillary atony is a terminal factor in grave diseases of diverse kinds. It produces clinical manifestations usually ascribed to intoxication. So far as the actual mechanism is concerned, many phenomena called toxic are essentially anoxic."

Further light has been shed on the renal mechanism of these conditions by the work of J. Trueta *et alii*.[4] These workers demonstrated the existence of two potential renal circulations, a greater and a lesser. The vessels of the greater pathway are those associated with cortical glomeruli and the channels of the lesser are associated with the juxtamedullary glomeruli.

In response usually to neurogenic or hormonal stimuli blood may pass almost exclusively through one or the other and frequently the cortical pathway may be bypassed altogether. Consequently a cortical anoxia as opposed to renal anoxia is postulated. One surgical application concerns the anuria occurring in the crush syndrome. In this condition, in response to peripheral nervous stimuli, renal vasospasm with diversion of circulation from renal cortex occurs. A pallid cortex with ischæmic glomeruli and congested medulla is seen at autopsy.

Changes in the renal circulation and especially a variable distribution of the blood flow between cortex and medulla reflect the operation of a normal vascular mechanism for maintaining the fluid balance of the body, and these workers suggest that these changes are effected via the autonomic nervous system by its direct action or as the result of the liberation of the hormones pituitrin and adrenaline. When the fluid balance of the body is grossly disturbed there is probably over-activity of this normal mechanism with resultant renal disturbance.

Bypassing of cortical glomeruli by the circulation → cortical anoxia → diminished glomerular filtration. Increased medullary circulation leads to increased tubular resorption, which again reduces urinary output.

The only progress then made in the understanding of pyloric stenosis, with azotæmia in the past twenty-five years has been a realization of the significance of the disturbed dynamics of the circulatory system and a better knowledge of the mechanics of the intrarenal circulation. Further doubt has been cast on the role played by unidentified toxins.

Summary.

1. Four cases of azotæmia following severe fluid loss from the upper gastro-intestinal tract are reported. Three cases followed pyloric stenosis and the other case followed gastro-enterostomy and vagotomy for a similar condition.

2. Disturbance of the dynamics of the circulatory system and of the intrarenal circulation appears to account for the outstanding features of the syndrome.

3. Further doubt is cast on the role played by toxins.

Acknowledgements.

I wish to thank Mr. K. W. Starr for his ever-ready help and advice, and for his permission to publish two of the cases.

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PENETRATING INJURY OF THE CRANIAL VAULT.[1]

By JOHN HUNTER and GILBERT PHILLIPS,
Sydney.

THE following case is reported as it clearly illustrates the severe intracranial disturbance which may succeed an apparently minor wound of the scalp, which has not been accompanied by signs of concussion or cerebral damage. It is now realized, particularly following experience of air raid and battle casualties, that the extent of the external wound to the head is no indication of the amount of trauma which has been caused to the brain. Penetrating injuries such as this are frequently not associated

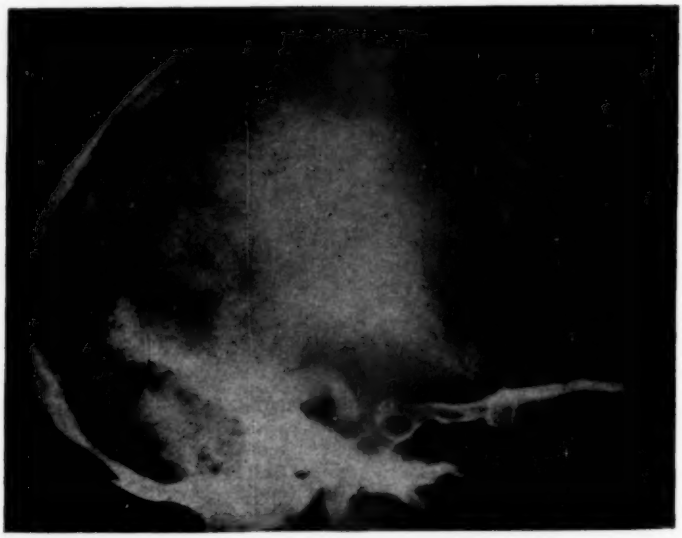

FIGURE I.

with any alteration of the conscious level, and too much emphasis cannot be placed on the adequate study of early radiographs in the interpretation of the nature and the extent of the intracranial damage.

This case will also illustrate the appropriate surgical technique in penetrating wounds of the brain and the use of electroencephalography in assessing the completeness of the surgical procedure performed.

Case History.

In February, 1946, Mr. McD., a labourer, aged twenty-nine years, sustained a head injury in the region of the vertex of the skull, caused by a falling wedge, whilst working in a caisson under conditions of increased atmospheric pressure. As he fell to the ground other tools struck his body, causing injury to the left side of his chest and pelvis. No loss of consciousness resulted, and following recovery from the initial shock the patient was able to stand and walk, but complained of pain in the chest on inspiration. He was hoisted from the caisson and taken to hospital, where X-ray examination revealed fractured ribs, but no osseous lesion in pelvis or skull. On examination of the head a small scalp laceration was noted in the region of the vertex; this was dressed and his chest was strapped. He was allowed home that evening. The next two weeks were passed uneventfully at home. The patient was not as

[1] Accepted for publication on November 20, 1947.

alert as usual, but nevertheless was able to perform light work. His only complaint was that of an occasional slight generalized headache.

Sixteen days after the original injury, however, there commenced to develop a progressive weakness of the right leg, associated with difficulty in controlling its movements and a tendency to drag the toes of the right foot whilst walking. These symptoms gradually became more marked and during the next few days were augmented by the onset of a similar weakness of the arm and hand on the right side. This loss of function was observed to spread in a progressive manner, commencing in the muscles of the upper part of the limb and extending to the fingers, thumb involvement being a terminal feature.

With these symptoms he returned to hospital. Further X-ray examinations were carried out and a provisional diagnosis of calcified subdural hæmatoma or, alternatively,

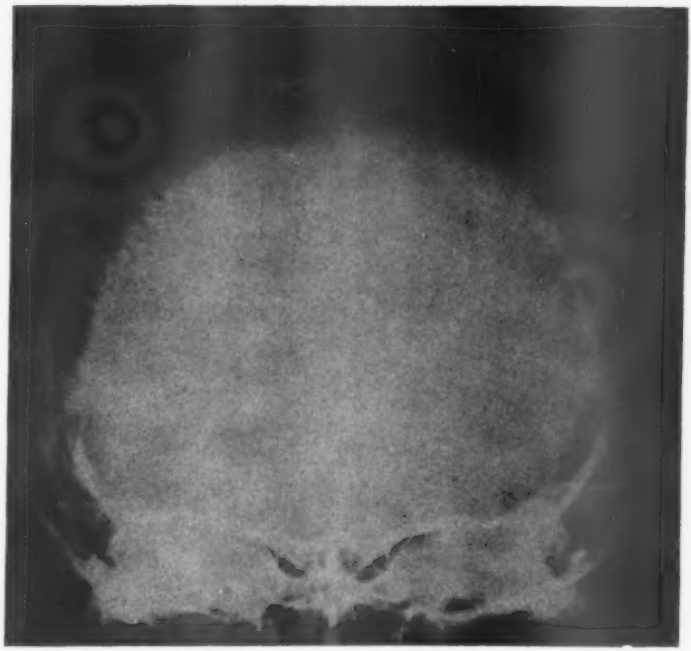

FIGURE II.

a depressed fracture of the skull was made. It was at this juncture that the patient was referred to the Department of Neurosurgery of the Royal Prince Alfred Hospital, approximately one month after his injury.

On his admission to the department there was no impairment of consciousness and no history of alteration of the mental state. Visual acuity was not impaired, and nausea, vomiting or disturbance of urinary function had not been manifest. The patient, however, still complained of a mild degree of headache. He appeared to be a sick man; his colour was an ashen grey, and he had apparently lost a considerable amount of weight. A right hemiparesis was present, more marked in the lower limb, where the main weakness was observed to be in the dorsiflexion of the ankle. The gait was characterized by circumduction of the right leg and a dragging of the toes of the right foot. Examination of the scalp revealed a small discharging sinus over the posterior portion of the left frontal bone, approximately 3·0 centimetres from the mid-line. Early papillœdema was observed in the left fundus and a doubtful upper motor neurone type of facial weakness on the right side. The absence of a right abdominal reflex was noted, together with an extensor plantar response on that side. Skin sensation was unimpaired.

Investigations and Progress.—Plain X-ray photographs of the skull (Figures I and II) showed a fracture of the left parietal bone near the vertex, with some

depression of many bony flakes. A blood leucocyte count of 11,000 white cells per cubic millimetre was reported and a culture growth from the discharging sinus was identified as hæmolytic *Staphylococcus aureus*. Lumbar puncture pressure was 200 millimetres of water. The cerebro-spinal fluid was clear. There was no alteration of cerebro-spinal fluid on biochemical or cytological examination.

FIGURE III.

Operation.—On March 25 a craniotomy was performed under local anæsthesia. The scalp laceration was completely excised in an elliptical manner, and directly beneath this a bony defect measuring 1·5 by 2·0 centimetres was found. On enlargement of this opening the dura was observed to have been lacerated and driven into the cortex. Beneath the skull defect a sinus tract led from the subdural space through the cortex downwards and forwards parallel to the superior sagittal sinus, and terminated in a loculus containing thick yellow pus. The subdural space also contained a localized collection of pus, being

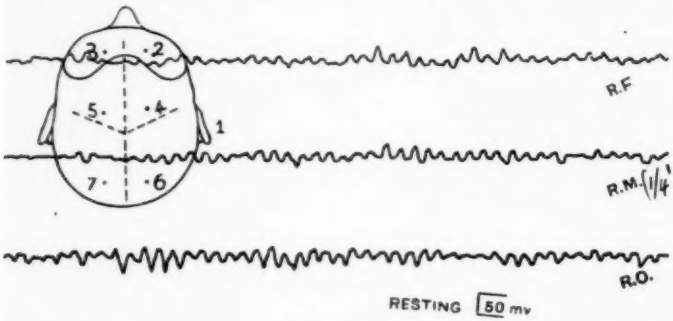

FIGURE IV.

walled off from the surrounding space by adhesions between bone, dura, arachnoid and cortex, and the subarachnoid space sealed off by adhesions around the lips of the cortical laceration.

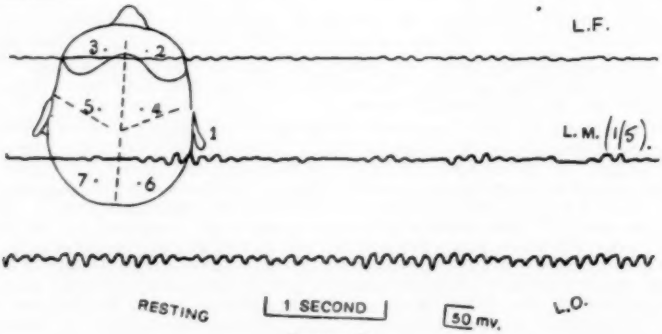

FIGURE V.

By means of suction the abscess was emptied and necrotic tissue from its walls was removed. Fifteen separate small spicules of bone (Figure III) were also removed from the region of the abscess and the surrounding cortex—the majority lying at a

depth of 3·0 to 4·0 centimetres from the surface. Penicillin and sulphanilamide powder were insufflated into the cavity and its tract, and the scalp was closed in layers without drainage.

Post-Operative Progress.—Immediately following operation the patient was referred to the electroencephalographic unit (unfortunately on this occasion an electrogram could not be arranged pre-operatively). The findings in this instance, recording on each side (Figure IV right, Figure V left), from the ear (indifferent electrode) and from the frontal, motor and occipital regions, was a normal α rhythm of 10·5 seconds, somewhat suppressed on the left side in all leads, but in particular from the frontal and motor placements. Significant, however, was the absence even after forty minutes of continuous recording, of any abnormal waves of cortical origin—in particular the slow δ activity characteristic of cerebral abscess—thus interpreted as a satisfactory and sufficiently radical removal of necrotic brain and damaged cortical tissue, and confirmed, to some degree, by a post-operative X-ray examination of the skull, which showed that only one small flake of bone had not been removed.

Clinically, on the day following operation a definite improvement in the right arm, hand and finger movements had occurred; the range of leg flexion movements had also increased. As the days passed, the gradual return to normal power and movement range was observed, hand movements recovering before arm movements and gross leg movements before those of the foot and toes.

On the fifth day there was very little residual paresis. The papillœdema was subsiding and a repeat electrogram was again within normal limits, and the suppressed α activity previously observed on the left side had returned almost to the same intensity as that of the right side.

Nineteen days after operation the patient was discharged from hospital, symptom free apart from a slight limp. When seen recently he was quite well and had returned to work.

Comment.

The following conclusions may be made:

1. A small wound in the scalp may be associated with a compound depressed fracture of the cranial vault, dural laceration and multiple indriven bone fragments in the brain.

2. This type of injury may occur in the absence of any signs of concussion or evidence of focal damage to the brain.

3. Indriven bone fragments frequently lead to the formation of a cerebral abscess.

4. Electroencephalography is a useful method for determining the adequacy of the surgical procedure and may disclose the presence of a persistent post-traumatic dysrhythmia, which in some cases is the precursor of traumatic epilepsy.

It is suggested that in the presence of any scalp wound overlying the cranial vault X-ray studies should be made at an early stage and the radiographs examined carefully for the presence of indriven bone fragments. If found, these constitute an indication for immediate operation, when the scalp wound should be enlarged, the edges of the bone defect removed, the dural opening enlarged, and *débridement* of the track carried out, all the bone fragments being removed. This should be checked radiographically. The track should then be insufflated with penicillin-sulphamezathine powder, the dura left open and the scalp wound closed. Under ideal conditions pre-operative and post-operative electroencephalographic studies should be carried out.

HYDATID CYST OF THE LEFT LOBE OF THE THYREOID.[1]

By V. M. COPPLESON,
Sydney.

THE patient was a married woman, A.W., aged forty-seven years, who lived for the first eighteen years of her life at Young and since then at Cootamundra, New South Wales. She had always kept dogs.

About one year before admission to hospital she noticed a lump on the left side of her neck and she had been intermittently hoarse for the previous eight months.

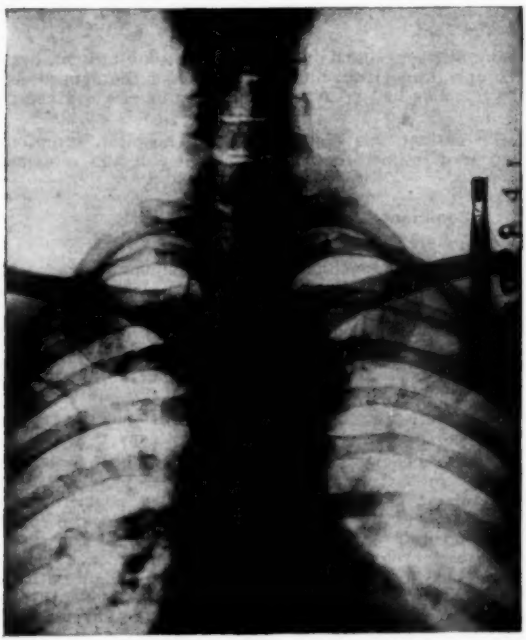

FIGURE I. Showing skiagram before operation.

She was admitted to Saint Vincent's General Hospital. Examination of her neck revealed a firm, hard swelling in the left lobe of her thyreoid. It was movable on deglutition. It was firmly fixed to the trachea. There was a marked deviation of the trachea to the right and examination of the larynx revealed left recurrent nerve paralysis.

At operation the isthmus of the thyreoid was divided. The left lobe of the thyreoid was found to be quite free on the outer side. It was firmly adherent to the trachea. Separation from the trachea showed that the trachea was part of the false ectocyst and it appeared to be at an early stage of perforating the trachea.

During the separation clear fluid characteristic of a hydatid escaped. The hydatid membrane was seen and immediately recognized. The left lobe of the thyreoid, including the cyst, was removed. The recurrent nerve was an immediate posterior relation. One hundred thousand units of penicillin were placed in the cavity and the wound was closed without drainage.

The patient made an uninterrupted recovery. There was considerable improvement of the hoarseness before she left hospital.

[1] Accepted for publication on May 17, 1948.

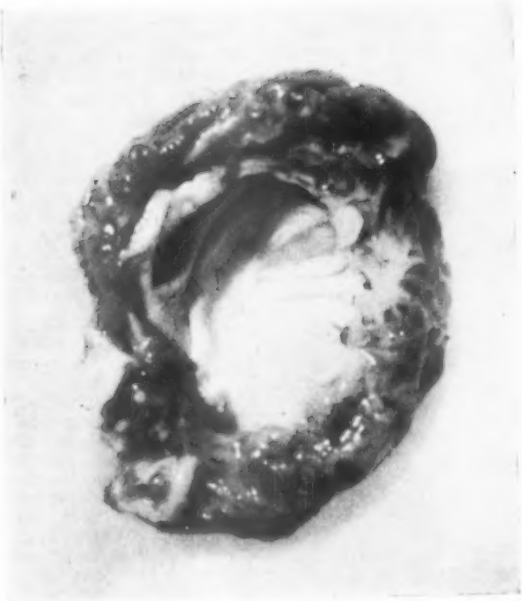

FIGURE II. Showing photograph of the cyst.

TWO UNUSUAL CASES OF INTUSSUSCEPTION OCCURRING IN CHILDREN, WITH A REVIEW OF THE LITERATURE.[1]

By DONALD HIPSLEY and ERIC GOULSTON,
Sydney.

INTUSSUSCEPTION is essentially a condition occurring in infancy and childhood for which often no attributable cause can be discovered. When the condition occurs in the adult some pathological condition can usually be demonstrated to explain its occurrence on a mechanical basis, whereas such conditions occur very rarely in the former age groups.

The following two cases are reported as illustrative of the varied symptoms and signs of intussusception occurring in children. Case I shows the remarkable degree of chronicity which can be present, and Case II the acuteness and rapid progression to a state of shock which can occur. In both cases bowel resection was necessary, in the former as an operation of election and in the latter as an operation of emergency.

Case Histories.

Case I.

A.McL., a male, aged three years and eight months, was first admitted to the Royal Alexandra Hospital for Children, Sydney, on November 2, 1947. There was nothing relevant in his past history and he had first complained of abdominal pains eight days previously. These pains were occurring frequently; they were colicky in nature and he would afterwards pass flatus. The mother stated that three to four days after the first attack of pain she had noticed a lump, which she demonstrated as being in the left hypochondrium. Previously the patient was admitted to the district hospital and given a bowel wash-out, after which it was stated that the lump disappeared. On November 2 he had another attack of abdominal pain, the lump was again palpable, and for the first time he passed some congealed blood *per rectum*, the bowels having been constipated for the previous two days. On examination the child did not appear at all ill and no abnormal physical signs were apparent. No tumour was detected on palpation of the abdomen, but on rectal examination there appeared to be a soft boggy mass high up in the rectum, and bright blood was present on the gloved finger. The child was then given a saline bowel wash-out, but the return was clear. Three days later, under "open" ether anæsthesia, a sigmoidoscope was passed for a distance of 17·0 centimetres, but no abnormality was detected. The possibility of Henoch-Schönlein's syndrome was considered, but nothing was found to support this diagnosis. The patient had remained very well whilst in hospital and arrangements were made for a barium enema after discharge from hospital. On November 15 the child was again admitted with the history that he had been having attacks of abdominal pain daily, which caused him to writhe and cry, pass flatus *per rectum* and then become languid. His bowels had been opened daily and there had

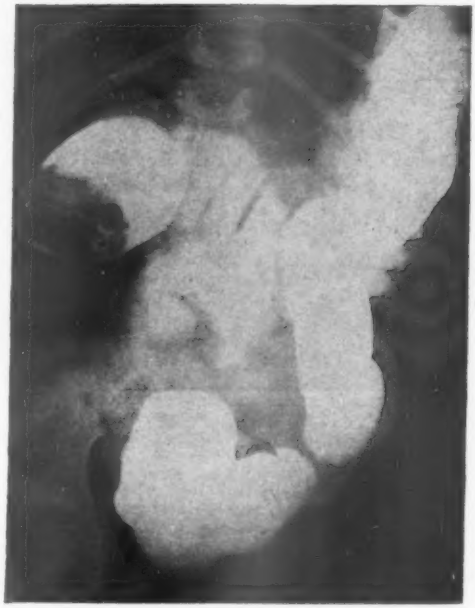

FIGURE I. Case I. Barium enema showing intussusception.

[1] Accepted for publication on June 15, 1948.

been no evidence of blood in the motions until the day prior to admission, when after an enema a small quantity of bright blood was recovered. The barium enema in the meantime had been carried out, which suggested intussusception (see Figure I).

On examination the child again appeared very well, but now a definite tumour was palpable in the epigastric region, approximately four inches by two inches in measurement. Under "open" ether anæsthesia the child was given a high saline solution bowel injection by the technique as described by P. L. Hipsley,[1] with the following result. The first return was clear fluid; the second return contained fæcal stained fluid, as did the third return. The tumour which was palpable prior to operation was not palpable after the high saline solution injection and it was assumed that the intussusception had been reduced successfully. When the child had regained consciousness from the anæsthetic he was given a drachm of charcoal by mouth, which was recovered eight hours later by a small saline rectal wash-out. The following day a barium meal and follow-through were performed, which indicated that the intussusception had recurred, but there was no obstruction to the passage of the meal (see Figure II). It was then decided that a laparotomy must be performed, and on the following day, under "open" ether anæsthesia the abdomen was opened through a lower paramedian incision. A definite caeco-colic intussusception causing partial intestinal obstruction was still present, with approximately two inches of tumour still unreduced, and the caecum and terminal ileum were markedly inflamed and œdematous. The reduction was easy and a caecopexy was performed in the hope of preventing further recurrence by fixing the caecum to the right iliac fossa by means of two silk sutures. The child made an uneventful recovery from the operation and was discharged from hospital ten days later, still with some thickening in the region of the caecum, but having normal daily bowel actions.

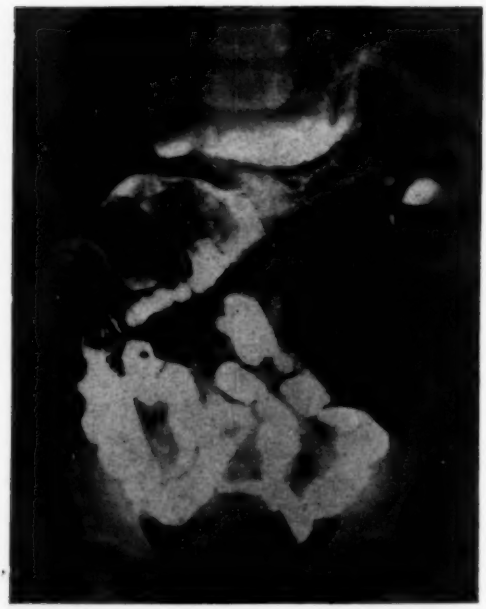

FIGURE II. Case I. Barium meal showing intussusception. There is no obstruction to the passage of the meal.

On December 18 the child was again admitted to hospital with the history that he had been suffering from diarrhœa for the previous four days with blood and mucus in the bowel motions for one day. There was no vomiting and again the patient did not appear to be particularly ill; but the abdomen was now quite distended and there was a large tumour palpable in the right iliac fossa, which was quite firm and tender and appeared to be inflammatory in nature. Expectant treatment was given with local heat to the abdominal tumour, and parenteral administration of penicillin, 10,000 units every three hours, was commenced, also sulphaguanidine in the dose of 0·5 gramme every four hours as a prophylactic measure. Within forty-eight hours the abdominal distension had decreased considerably and the tumour was subsiding. In view of the recurrent nature of the condition a Mantoux test was performed to exclude tuberculosis; this gave a negative result with a dilution of 1 in 1,000. In view of the chronicity of the symptoms, the constant presence of a mass and the radiological findings, on February 4, 1948, a second laparotomy was performed and it was found that the inflammatory reaction had now almost completely subsided. There appeared to be a tumour in the terminal part of the ileum which on palpation appeared to be quite discrete, and after an opening had been made into the ileum this tumour was thought to be a sessile polypus. It was considered that this tumour had been acting as a mechanical factor in causing the chronic intussusception, and it was decided that resection was necessary to prevent further recurrence. Six inches of the terminal part of the ileum and two inches of caecum were resected and a side-to-side anastomosis was performed. Twenty thousand units of crystalline penicillin powder were placed in the peritoneal cavity. Throughout the operation a transfusion of whole blood

followed by serum was given. Sulphapyridine ("M & B 693"), 0·5 gramme, was given intravenously every four hours for twenty-four hours, after which sulphaguanidine was again commenced (0·5 gramme every four hours) and continued for a further seven days. The child made an uneventful recovery and has remained well up to the time of this publication.

Pathology.—Subsequent pathological examination of the resected portion of bowel showed a marked lymphoid hyperplasia, so that the bowel wall in this part was thickened. The mucosa was nodular and corrugated, and where the lymphoid masses approached the epithelial surface they appeared as pale areas against the dark background of congestion. These points will be appreciated by reference to Figure III, surrounding the region marked *c*. Such lymphoid tissue is often prominent in the bowel at the age period of this patient, but it is not found to the extent seen in this case except under pathological conditions.

Case II.

T.S., a male, aged eight years, had had one attack of abdominal colic six months prior to his admission to the Royal Alexandra Hospital for Children, Sydney, on January 22, 1948, and had also been diagnosed as suffering from *prolapsus ani* on this occasion, but we presume that the lesion was a rectal polypus which was removed subsequently. He had a fourteen-hour history of severe abdominal pain which had persisted without any decrease in severity throughout the day. An hour after the onset of pain he commenced to vomit and he had been vomiting and dry retching throughout the day. He was unable to keep down any fluids, and the vomitus, which was not offensive, consisted of clear fluid only.

FIGURE III. Case I. Pathological specimen of resected portion of bowel. *a* = caecum, *b* = ileum, *c* = nodules of hyperplasia.

On examination he was extremely apprehensive and pale, with cyanosis of the lips, and he looked obviously shocked. The pulse volume was weak and the rate was 110 per minute. The abdomen was distended, a large irregular tumour being visible in the left hypochondrium. No peristalsis was visible. There was generalized abdominal tenderness and the tumour was hard but not fluctuant and obviously caused him much pain when palpated. Borborygmi were audible, but not excessive, on auscultation of the abdomen. A plain skiagram of the abdomen was taken before the patient's admission to the ward; this showed a dilatation of the proximal two-thirds of the colon due to gas and possibly a fluid level, but it was impossible to locate the site of the obstruction.

On his admission to the ward restorative measures were instituted, consisting of intravenous serum therapy and continuous gastric suction by means of the Wangensteen's apparatus as modified by Gill.[2] A diagnosis of intestinal obstruction was made, the history indicating that the obstruction was probably high in the jejunum. After the administration of 400 cubic centimetres of serum it was considered that the child's condition had improved sufficiently for laparotomy to be performed. He was taken to the theatre and the serum was replaced by a continuous intravenous drip of whole blood. Under ether and oxygen anæsthesia the abdomen was opened through a left upper paramedian incision. On the opening of the abdomen a large tumour presented, which at first was difficult to recognize as being an intussusception in the jejunum, as there was some degree of torsion present (see Figure IV). The apex of the tumour was about 12 inches from the end of the duodenum. It was found to be impossible to reduce the intussusception and so the tumour was resected in its entirety. About three inches from the distal end of the tumour there was an indurated area in the bowel wall which was included in the resection. Both stumps were closed and a side-to-side anastomosis was performed. Two hundred thousand units of crystalline penicillin powder were placed in the peritoneal cavity and the abdomen was closed without

drainage. Following the operation gastric suction was continued for forty-eight hours and intranasal oxygen for twenty-four hours. The intravenous administration of N/5 saline solution with 5% glucose was continued for four days and 1·0 gramme of soluble sulphapyridine ("M & B 693") was put into the tube every four hours for this period.

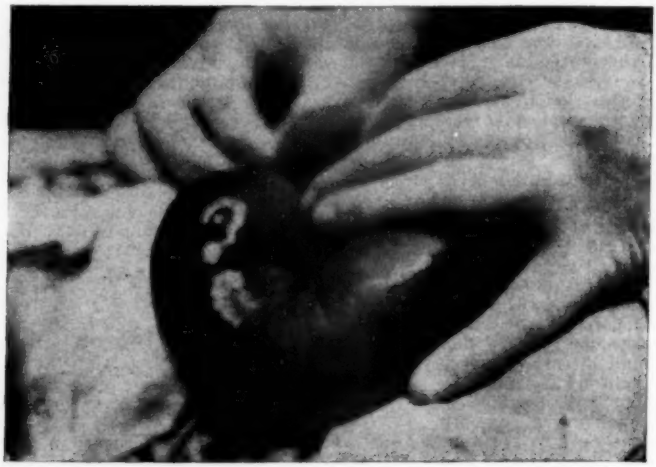

FIGURE IV. Case II. At operation, a large tumour in the jejunum which consisted of an irreducible intussusception which had undergone some degree of torsion.

Penicillin, 30,000 units, was given intramuscularly every three hours for two days following the operation and then 100,000 units every eight hours for a further eight days, and sulphadiazine was given by mouth every four hours for the same period.

FIGURE V. Case II. Pathological specimen showing pedunculated polypus at proximal end which had formed the apex of the intussusception.

Apart from some mild delirium on the day following operation, requiring sedation with *Haustus Paraldehydi*, the child made an uneventful recovery. On the twentieth day following operation a pedunculated polypus was removed from the rectum. The child has since reported to us and is perfectly well.

Pathology.—Even in the resected specimen it was impossible to reduce the intussusception until a small nick had been made in the mesentery at the neck. This released approximately eight to ten ounces of thin straw-coloured fluid and then the whole intussusception was able to be reduced. The specimen then measured two feet three inches long and one and a quarter inches thick. There were two lobulatd tumours, one at each end. The smaller tumour, being the one which had formed the apex of the intussusception, had a pedicle 2·0 centimetres long and was obviously inflamed and congested (see Figure V). The larger tumour, which was situated at the distal end, was practically sessile (see Figure VI). Note the two tumours have been bisected for microscopic examination, and it is the cut surfaces which are shown in the photographs. Also it will be noticed that at the base of the sessile tumour in Figure VI a section of the bowel has been removed in order to investigate any evidence of infiltration. There was nodular thickening of the wall of the bowel at the point of attachment of the sessile polypus and there was an increase in thickness in the wall of the entire segment of bowel, consisting largely of muscular hypertrophy, evidence of chronic intestinal obstruction. The bowel lumen was also filled with dark semi-fluid contents and the mucosa, which was congested in all parts, was dark black to red throughout the specimen, evidence of acute intestinal obstruction.

Microscopically both tumours were essentially similar in structure, though inflammation and congestion were much more marked in the smaller one, which had been situated at the apex of the intussusception. They were both adenomatous polypi and there was no evidence of any infiltration of the bowel around the growths by cells or mucus.

FIGURE VI. Case II. Pathological specimen showing sessile polypus at distal end.

Comment.

At first it was thought that these two cases had a similar ætiology, that is, a polypoid condition of the small intestine, but on pathological examination of the specimen from Case I it was found that the tumour which had been palpable in the terminal part of the ileum at the time of operation, was due purely to lymphoid hyperplasia. It can be argued that the hyperplasia may have been the result and not the cause of the intussusception, but we feel that the polypoidal area played its part as the mechanical factor in this case. There can be no doubt that in Case II a polypus acted as a mechanical factor in producing the intussusception, for, situated at the apex of the resected length of bowel, there was a pedunculated polypus which showed considerable signs of congestion.

Discussion with a Review of the Literature.

It is generally admitted that in 90% to 95% of cases of childhood intussusception the ætiological agent is still unknown. Many authors, particularly Walton,[3] Hipsley,[4] and Clubbe,[5] have ascribed as a cause hypertrophy of Peyer's patches of lymphoid tissue in the terminal part of the ileum. It is interesting to note that forty-one years ago P. L. Hipsley made the following statement and published a photograph of a case which we have been kindly permitted to reproduce in this article[4] (see Figure VII): "If such patches were hypertrophied or swollen, they might act in the same way as a polypus."

Our Case I was apparently such a one, and at operation the tumour demonstrated in the terminal part of the ileum was mistaken for a polypus. In considering the ætiology of intussusception it is important, if possible, to determine first the exact position of the primary invagination, and in the enteric variety, according to Corner,[6] the most common position is in the last inch of the ileum; and, moreover, he asserts that a lateral and not a circular origin is most common. Clubbe agrees with Corner in his findings and states that "in intussusception beginning in the ileum the first infolding seems frequently to start at the side of the bowel and always opposite the mesenteric attachment". He also says that "cases which are primarily enteric generally start a few inches from the caecum". This is the position also of Peyer's patches. Hipsley also comments, in support of this theory of lymphoid hypertrophy or hyperplasia having an important bearing on the occurrence of intussusception in children and infants, on the following facts: (i) Peyer's patches stand in greater contrast to the thin bowel wall in children than adults. (ii) Intussusception is most commonly met with just at that period of life in which the lymphoid tissue occurs in the bowel in the greatest quantity, that is, in

FIGURE VII. Pathological specimen of case reported by P. L. Hipsley (1907). *a* = caecum, *b* = ileum, *c* = hypertrophied Peyer's patch. (Reproduced from *The Australasian Medical Gazette.*)

infancy and childhood. Also it is extremely rare to meet with the condition in infants under the age of one month, lymphoid tissue being developed only in the caecum and caecal appendix during the first week after birth. (iii) In support of a statement in Osler's "Textbook of Medicine" that in children and infants Peyer's patches become swollen from very slight causes, whereas in adults, apart from enteric fever, they are rarely affected.

Hipsley[4] examined the bowel in thirty patients suffering from different diseases, the majority from gastro-enteritis which did not show any hypertrophy. The ages of the patients ranged from three to eighteen months. The cases which showed the most marked degree of hypertrophy of Peyer's patches were two cases of intussusception, one of which has been referred to in the present article. In these two cases the hypertrophy was more marked in the terminal portion of the ileum, and although the author admits that this may have been only a secondary condition following on the congestion due to the intussusception, the hypertrophy, to him, appeared to be out of all proportion to the congestion of the bowel wall. He also made reference to cases of pertussis, of tuberculous disease of the intestine and of typhoid fever in a child of three years, all of which had an intussusception of the bowel as a complication. Ladd and Gross[7] found only seven cases in which intussusception had occurred as a sequel to dysentery or infectious diarrhœa in a series of 372 cases reported by them.

Mechanical abnormalities are often the cause of intussusception occurring in the adult, but such factors are found in the minority in the infancy and childhood groups. In the series of 484 cases reported by Ladd and Gross[8] polypus was found as a cause in less than 1%, and a definite attributable cause could be found in only 5% of the cases. These authors also advise that removal of a Meckel's diverticulum or other cause of intussusception be deferred if possible for a second operation (ten days or two weeks later), when the circulation will have returned to normal and the peritoneal cavity will better withstand any soiling which may occur with any operative procedure.

It is interesting to attempt to correlate the age incidence with these cases in which intussusception occurs and an attributable cause can be found. We are unable to ascertain the age incidence of the patients reported by Ladd and Gross. However, it would appear from a study of the age incidence of polypi of the bowel occurring in children, as observed by these authors, that they were in the older age groups. Ladd and Gross found that polypi of the bowel most frequently occurred in children of two to eight years and that there was a peak of incidence in children of four to five years, whereas it was rarely encountered in the first year of life. Recently Dennis[9] has reported a series of eight cases of intussusception in children in which a resection was necessary. Except for one patient, aged seven and a half months, in whom a Meckel's diverticulum was discovered at the apex of the intussusception, in all the patients under two years of age no attributable cause was detected. In one of his patients, aged forty-three months, he demonstrated a polypoid hyperplasia of a Peyer's patch about 3·0 centimetres from the ileo-caecal junction. This case appears to be remarkably similar to our Case I, except that the duration of symptoms appears to be somewhat shorter and that the resection was performed only five days after the original laparotomy. Another of the cases reported by Dennis was similar to our Case II, the patient being a boy, aged nine years, who was found to have a high ileo-ileal intussusception and a pedunculated polypus was demonstrated at the apex of the intussusception. Dickson[10] reports a case of a girl, aged twenty-two years, who had multiple adenomata of the jejunum. She had first had an intussusception at the age of seven years and later at the ages of twenty-two and twenty-six years, laparotomy being performed in each instance. The interesting features of the case were that she had episodes of abdominal pain throughout and that the offending polypi were removed by simple incision, for in each instance the intussusception was reducible and the bowel viable. On the third occasion the intussusception had been reduced prior to laparotomy, presumably by the external manipulation, but there was definite evidence to show where the intussusception had occurred.

Wardill[11] stresses the importance of making a careful examination of the bowel in all cases of intussusception occurring in children over the age of two years, for neglect of this precaution may lead to recurrence. He also points out that the polypus need not necessarily be placed at the apex, but that by lying free in the lumen it can act as a forign body and so bring about an intussusception. In both our cases the offending tumour was situated at the apex of the intussusception.

Fiske[12] reports a case of multiple adenomatous polypi occurring in a female child, aged ten years, who had always been undernourished and had lived almost exclusively on a milk diet. This child was subjected to several operations before the actual pathology of the condition was recognized, and then only because an intussusception developed which had to be resected. He considers that the relationship between intussusception and tumours or other gross causative pathological conditions cannot be too strongly emphasized, and quotes cases reported by Oughertson and Cheever,[13] Joyce,[14] and Haggard and Floyd,[15] in all of which several operations were performed

before the true pathological condition was recognized and corrected. He also states that of the benign tumours occurring in the small intestine, adenoma is the most common, and that it is generally stated that they rarely occur in the jejunum. Raiford[16] found only one of his fifteen cases of adenoma arising in the jejunum, while three of the eleven cases reported by Rankin and Newell[17] were jejunal in origin. Joyce's case was also in the jejunum. Other cases of polyposis of the small intestine causing intussusception have been reported, but they have usually been in young adults. Somerville-Large[18] has reported a case of a boy, aged seventeen years, with double intussusception associated with polyposis of the jejunum, this being similar to our case in that the onset was acute and resection had to be performed. Beales and Frankel[19] also report a case in an older patient, a woman aged thirty years, who had a history of attacks of upper abdominal pain for thirteen years prior to diagnosis and had repeated investigations of the gastro-intestinal tract which gave negative results, and was consequently labelled as neurotic. Our patient had had symptoms for six months, and if acute intestinal obstruction had not set in could have possibly been subjected to a similar series of investigations before a diagnosis was made. Yet a similar case to our Case II is reported in the case records of the Massachusetts General Hospital.[20] It is that of an unmarried nineteen-year-old girl who was apparently well until eight hours before admission to hospital and at operation was found to be suffering from a double ileo-ileal intussusception with volvulus led by an adenomatous polypus, the bowel having to be resected.

Certainly it would appear from a review of the literature that intussusception occurring in children, in which an ætiological cause can be demonstrated, does tend to occur in the older age groups, although in the five cases reported by Koster,[21] presumably due to adenomata, three cases were in infants four, six and sixteen months of age respectively.

Henoch's purpura was considered at one stage in the diagnosis in Case I. Wolfsohn[22] reports a case of intussusception and purpura, and indicates that this is a comparatively rare combination, as there appear to have been only nineteen similar cases reported in the literature since that reported by Vierhuff (1893).[23] Whitby and Britton[24] point out that although there is usually a combination of symptoms and signs, purpura of the skin with abdominal and joint pains, and although Henoch's purpura with intussusception does occur, it is diagnosed more frequently than it is found. They also point out that there is no diminution in the platelets in this condition. The results of blood examination in our case were normal in all respects.

In our Case II, due to polypus, a second polypus, sessile in nature, was removed along with the resected section of bowel. No other polypus was detected at operation, but during the convalescence another pedunculated polypus was removed from the rectum. Sigmoidoscopic examination did not suggest to us that the child was suffering from *polyposis intestini*, for apart from the polypus in the rectum there was no other evidence of polyposis or abnormality of the lower part of the colon, this being excluded by a barium enema given by the "Reduction Density Technique" of Dr. Norman P. Henderson as described by Gabriel.[25] The condition of *polyposis intestini*, as pointed out by Dukes,[26] is highly malignant and bears a definite familial tendency. Both parents were questioned in an endeavour to trace any evidence of cancer, but none could be found. Gabriel states that only rarely has polyposis been noted in the first decade.

Summary.

1. Two unusual cases of intussusception in childhood are presented, one illustrating a chronic variety due to polypoidal-like hyperplasia of the lower part of the ileum, the other an acute intussusception due to polypi of the upper part of the jejunum.

2. It is felt that operative methods of bowel fixation to prevent recurrence of intussusception are unnecessary and unlikely to be successful.

3. It is worth while to make a careful examination of the bowel in all cases of intussusception, especially in children over two years, as small tumours within the intestinal wall in an œdematous bowel may be easily overlooked.

4. Ætiology of intussusception is discussed with reference to lymphoid hyperplasia in the terminal part of the ileum.

5. Age incidence is discussed.

6. A review of the relevant literature is given.

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Abstracts from Current Literature.

[In this column will be published short résumés of articles likely to be of practical value from Journals published in other countries and not readily accessible to surgeons in Australia and New Zealand.]

SULPHASUXIDINE AND STREPTOMYCIN IN BOWEL TREATMENT.

Edgar J. Poth, Joseph P. McNeill, Louis J. Manhoff, junior, Walter B. King and John G. Sinclair: "The Healing of Bowel as Influenced by Sulfasuxidine and Streptomycin", *Surgery, Gynecology and Obstetrics*, Volume lxxxvi, Number 6, June, 1948, page 641.

THIS article records experiments on dogs designed to show the worth of sulphasuxidine and streptomycin in both open and "closed aseptic" anastomoses of the colon of dogs. Sixteen control animals were used in a total of 44 animals.

In open anastomoses a continuous through-and-through number 00 chromicized catgut suture was used to form the anastomosis, and a single layer of through-and-through chromicized catgut with an outer row of interrupted fine black silk sutures was used in the aseptic technique.

As the result of these experiments they found that in the control dogs with an anastomosis without sulphasuxidine or sulphathalidine there was a high percentage of wound infection or peritonitis and three perforations and three deaths, but with the closed technique the result was better than with the open technique.

When the dogs had been given streptomycin and sulphasuxidine the end results were uniformly good, whether open or closed technique was used. The value of sulphasuxidine was shown, but streptomycin used orally failed to affect bacterial flora and the results were no different from when sulphasuxidine was given alone.

J. DEVINE.

PLASTIC REPAIR OF FACIAL PARALYSIS.

Paul W. Greeley, M.D.: "Plastic Surgical Repair of Facial Paralysis", *Archives of Surgery*, Volume lvi, Number 2, February, 1948, pages 132-137.

DR. GREELEY, of the Division of Plastic Surgery, University of Illinois, discusses in this article the various methods and results obtained in the treatment of facial nerve injuries. In the selection of cases he advises that surgical repair of some sort should be done if considerable spontaneous return of function has not become manifest within three to four months after injury. Before plastic operation is decided upon he advises that in all cases a neurosurgical consultation should be held. He classifies operations for the correction of facial paralysis into four groups: (*a*) neurosurgical repair, (*b*) mechanical support with *fascia lata*, (*c*) transplants of temporal and masseter muscle, (*d*) combination of mechanical support with transplants of fascia and muscle.

The author reviews the various operations used in each of these groups, and in particular the modern modifications of the original Kirschner technique using autogenous *fascia lata*. These modifications involve, firstly, the point of insertion of the lower end of the fascial strip and, secondly, the attachment of the proximal end. He points out that better support of the lower lip is obtained if the terminal end of the fascia is carried completely across the lower lip beneath the vermilion border to the opposite or normal angle of the mouth, where it is sutured to the *orbicularis oris* muscle. So far as the proximal end of the fascia is concerned, it should be fixed to the temporalis muscle instead of to the temporalis fascia. He points out that the activation of the fascia so obtained is variable and has limitations because of the length of fascia involved. Improvement in the activation of the fascial sling is obtained by the use of muscle pedicles following the work of Gillies, Sheehan, Bruner, Adams and Owens. For the mouth, pedicles can be turned down from the temporalis or masseter; for the eye, two pedicles from the temporalis, extra length being obtained as necessary by the use of *fascia lata* strips.

The author points out that for success to follow muscle transplant operations full cooperation of an intelligent patient is essential.

A bibliography of the various operations is provided.

K. F. RUSSELL.

TRANSPLANTATION OF LEVATOR MUSCLES IN REPAIR OF COMPLETE TEAR AND RECTO-VAGINAL FISTULA.

Axal Ingelman-Sundberg: "Transplantation of the Levator Muscles in the Repair of Complete Tear and Rectovaginal Fistula", *Acta chirurgica Scandinavica*, Volume xcvi, Number 4, page 313.

THE author describes a technique in the operation for the cure of recto-vaginal fistulæ by the use of the levators in a new manner. He points out that one of the reasons for failure is that often healthy tissue is not available to interpose between the sutured rectal and vaginal walls. The usual method is to suture the medial edges of the two levators together, but this involves a certain amount of tension.

The author's operation is to divide the pubo-coccygeus muscles approximately in the middle on each side as they surround the vagina. The two posterior portions are then sutured over one another in front of the suture line in the anterior wall of the rectum, thus providing a healthy muscular barricade between the rectal and vaginal walls. The advantage claimed is that the levators (pubo-coccygeus) are sutured together without any tension and, secondly, that an excellent covering of the rectal suture is obtained.

The two anterior portions of the pubo-coccygeus muscles are used to elevate the bladder neck and uterus in the following manner. An incision is made in the anterior wall and a clamp passed through it and around the sides of the vagina, and the cut ends of the anterior portions of the pubo-coccygeus muscles are withdrawn. They are sutured to each other and both sutured to the cervix.

The author records one case with complete cure after two previous unsuccessful operations.

FRANCIS J. HAYDEN.

CANCER OF THE ŒSOPHAGUS AND GASTRIC CARDIA.

George T. Pack, M.D.: "Introduction to Symposium on Cancer of the Esophagus and Gastric Cardia", *Surgery*, June 23, 1948.

IN an introduction to a symposium on cancer of the œsophagus and gastric cardia George T. Pack reviews briefly the history of the surgery of the œsophagus and upper part of the stomach. He quotes illuminating figures on the incidence of these conditions which may not be appreciated by many surgeons. Carcinoma of the cardiac segment of the stomach (6%) and carcinoma of the œsophagus (4%) are responsible for 10% of all carcinoma of the alimentary canal. In other words, transthoracic œsophago-gastrectomy now, for the first time, gives 10% of patients who have carcinoma of the gastro-intestinal tract some prospect of cure.

Another point of some interest is that carcinoma of the œsophagus is a highly malignant tumour, a statement that is contrary to many contemporary opinions. Metastases often occur early and widely.

Radiotherapy is only of palliative value, although a new method of rotatory irradiation devised by Nielsen, of the Copenhagen Radium Centre, does give some hope of improvement by permitting larger doses to be given before cutaneous reaction occurs.

GRAYTON BROWN.

PROGRESS IN SURGICAL TREATMENT OF CARCINOMA OF THE ŒSOPHAGUS AND UPPER PART OF THE STOMACH.

John H. Garloch, M.D.: "Progress in the Surgical Treatment of Carcinoma of the Esophagus and Upper Stomach", *Surgery*, June 23, 1948.

GARLOCH gives his impressions of progress in the surgical treatment of carcinoma of the œsophagus and upper part of the stomach after an operative experience of at least 250 cases.

The study of the early pathological changes has been possible only with the recent increase in operative excisions. Cardiac segment carcinoma may spread a considerable distance up the submucous layer of the œsophagus. Lymph nodes in the mediastinum may be extensively involved in early cases. Squamous-cell carcinomata of the œsophagus do not spread downwards past the œsophago-gastric junction. However, peripheral spread may be early and cause inoperability. There may be spread to lymph nodes far removed from the growth, such as in the lower cervical or pancreatic nodes.

The progressive improvement in operative management has been due to (i) intra-tracheal gas-oxygen-ether anæsthesia, (ii) the appreciation of the height to which the stomach may be taken in the thorax and even the lower cervical region, (iii) combined abdomino-thoracic incision.

Important details of the operation which may be of interest are: (*a*) left phrenic nerve is crushed above the diaphragh, (*b*) no clamps are used, (*c*) interrupted silk

sutures for anastomoses, (*d*) slight telescoping of suture line, (*e*) careful anchoring of diaphragm to stomach to prevent herniation, (*f*) no indwelling tubes post-operatively across suture line, (*g*) underwater drainage for the pleural cavity.

The author finishes with a plea for a more accurate classification of operative results by surgeons, indicating particularly those excisions carried out for palliative reasons.

GRAYTON BROWN.

SURGICAL TREATMENT OF ESSENTIAL HYPERTENSION.

Leif Efskind: "Surgical Treatment of Essential Hypertension", *Acta chirurgica Scandinavica*, Volume xcvi, Number 5, 1948, page 393.

LEIF EFSKIND describes the results of a modified Smithwick operation carried out on forty-one patients suffering from essential hypertension. The patients have been followed up for from one to five years. The operative mortality was nil, but two patients died during the period of observation. The age group was between thirty-five and fifty-three years, and all subjects on admission to hospital had blood pressures of over 200 millimetres of mercury, associated with X-ray and electrocardiographic evidence of hypertrophy of the left ventricle, but without signs of markedly reduced renal function.

The first step at operation was to perform renal biopsy and if the renal capsule was thickened decapsulation was carried out. The author concludes that if the biopsy reveals extensive thickening of renal arterioles, exudate in Bowman's capsule or regressive changes of the epithelium of the tubules, little reduction of blood pressure results. But if the patient had severe subjective symptoms these may be considerably improved nevertheless. The further steps involve a supradiaphragmatic and infradiaphragmatic resection of the splanchnic nerves and removal of the sympathetic ganglia from the ninth thoracic to the second lumbar level together with the cœliac ganglion.

In eight cases the blood pressure returned to normal levels. Sixteen cases showed a fall of blood pressure of between 60 and 110 millimetres of mercury, and seven cases between 30 and 55 millimetres of mercury. The remaining ten cases showed no reduction, but only three of them had a post-operative rise in blood pressure.

Patients with labile blood pressure, as indicated by response to rest and by the effects of the administration of sodium nitrate or of amytal, gave the most satisfactory post-operative results. Contraindications to operation are a fixed blood pressure and a distinct bilateral renal arteriosclerosis as revealed by biopsy.

In patients whose blood pressure falls to normal or is greatly reduced, the hypertrophied hearts can be shown to return towards a normal size within two months.

If the effect of sympathectomy in cases is disappointing as regards the fall of blood pressure, the author points out that the disease from the patient's point of view is often not to be measured in millimetres of mercury.

ROBERT OFFICER.

MARROW-NAILING OF RECENT FRACTURES, PSEUDARTHROSIS AND BONE PLASTIC.

Anders Westerborn: *Annals of Surgery*, Volume cxxvii, 1948, page 577.

WRITING from the Sahlgrenska Hospital, Gothenberg, Sweden, Westerborn describes his experiences with the Küntscher nail in the treatment of a series of 100 cases, including recent fractures, pseudarthrosis and bone plastic operations. The principle involved is the insertion of an intramedullary splint which spans the fracture site and allows early mobilization and, in the case of the femur, weight-bearing.

The nail is inserted through a small hole drilled in the cortex of the bone, a considerable distance from the site of the fracture. As a rule, in fresh fractures closed reduction is the method of choice, the nail being inserted under fluoroscopic control or preferably by being repeatedly checked by several X-ray examinations. The use of a Kirschner wire as a guide is also suggested to prevent malplacement of the nail. Occasionally open reduction is necessary, but with the use of the antibiotics the risk of untoward accidents is very slight.

The actual nail is of rustless steel and is V-shaped in cross-section, and thus does not fill the whole medullary cavity and causes very little damage to the bone marrow or endosteum. Nevertheless, if the right-sized nail is chosen, very firm fixation of the fragments is obtained.

In any operative procedure using the Küntscher nail it is necessary to have several nails of different widths and lengths at hand, and also an apparatus to remove the nail if it happens to lie in a wrong position.

As a general rule the nail is removed after the fracture has united. A technical point of extreme importance is that one chooses a nail that fits well into the marrow

cavity and to ascertain that the latter is of fairly even width. This is the case in the cavity of the femur and to a certain extent in the radius and ulna, but not in the humerus or tibia. Thus the best results are obtained in nailing femoral fractures. The size of the marrow cavity, and thus the correct nail to use, can usually be gauged accurately from X ray photographs.

A series of case histories illustrating the use if the nail in recent fractures is appended.

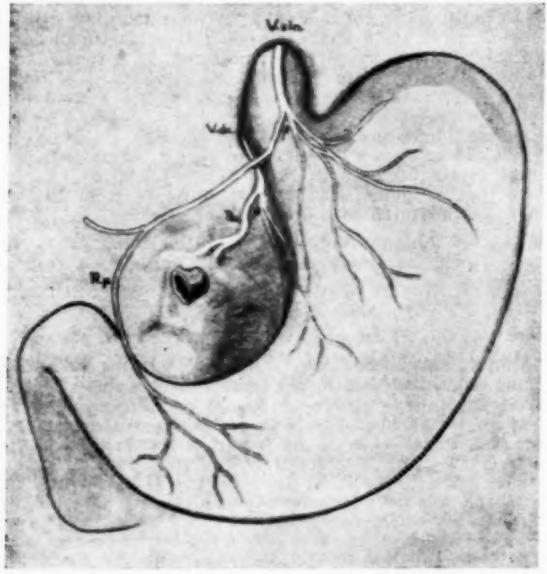
According to Westerborn, the advantages of medullary nailing, particularly in femoral fractures, include a shortened stay in bed, reduction of pain and other subjective symptoms, early ambulation and more speedy rehabilitation. Certain risks are involved, those mentioned being marrow destruction, fat embolism and osteomyelitis. According to the author, however, these occurrences are distinct rarities. The beneficial results of the use of the Küntscher nail in the treatment of pseudarthrosis is discussed with illustrative case histories. It is pointed out, however, that it is a more difficult procedure in these cases, because the sclerosis in the ends of the bones offers powerful resistance and may even make it impossible to insert the nail. Finally the use of the nail as a fixative agent following operation for bone lengthening or shortening is briefly described along with some case histories.

F. J. CAHILL.

ABDOMINAL VAGOTOMY.

Curt Franksson: "Selective Abdominal Vagotomy", *Acta chirurgica Scandinavica*, Volume xcvi, Number 5, 1948, page 409.

MANY believe that hydrochloric acid is mainly responsible for the development of peptic ulcer, and one of the latest means of decreasing acidity is vagotomy. Complete division of the vagi, whether above or below the diaphragm, results in interruption of vagal influx to all abdominal viscera.



Franksson describes the anatomical distribution of the vagus nerves and recommends section only of those branches innervating the acid-producing portion of the stomach.

Two to three centimetres below the diaphragm the left vagus nerve gives a branch which travels through the lesser omentum to the portal fissure, giving a branch to the duodenum and pylorus on the way. The remaining portion of the nerve supplies the stomach.

The right vagus nerve supplies the stomach also, but a large branch arises from it three to four centimetres below the diaphragm. This branch conveys impulses to the remainder of the alimentary tract by way of the cœliac plexus.

The aim of the selective abdominal vagotomy is to cut branches of both nerves destined for supply of the stomach, while sparing the large branch of the right vagus passing to the cœliac plexus and the branch of the left nerves passing to the liver, duodenum and pylorus. The operation is performed as follows. The left lobe of the liver is mobilized. The vagus nerves are dissected from the œsophagus and stretched by means of the loops of catgut placed around them. By this means the branches are rendered taut and can be freed by blunt dissection. The branches destined for the stomach only are sectioned.

Six cases have been thus treated and remarkable reduction of the acid levels, as shown by the insulin test, have resulted. The ulcers in five cases healed rapidly.

ROBERT OFFICER.

RESECTION OF THE INFERIOR VENA CAVA.

R. Leriche: *"Nécessité de l'intervention immédiate dans les phlébites de le veine cave inférieure: Un cas de résection segmentaire de la veine cave"*, *Lyon chirurgical*, Volume xlii, 1947, page 385.

THE evolution of phlebitis in the lower limbs as at present understood appears to be conditioned by two factors, of which one is the process of coagulation and the other spasm of the vessel immediately above it. Judging by the comparative effects of the two main methods of treatment, sympathetic block for spasm and the use of anti-coagulants for clotting, the spasmodic factor would seem to be the more important of the two. Once, however, actual mechanical blockage of the vessel lumen has occurred, neither of these lines of attack is effective. In respect to the patient's future welfare, more danger seems to lie in the possibility of extension of the clot to the *vena cava* than in possibilities of its detachment. It is Leriche's opinion that thrombosis of the *vena cava* is never primary, but always secondary to an original focus in one or other common iliac vein, most usually the left. Unfortunately the disease does not remain endovenous, but produces associated changes which greatly add to the difficulties of surgical treatment. One such effect is the involvement of lymphatic vessels and glands, which form a dense sclerotic mass round the vessels and complicate matters by adding lymphatic to venous stasis.

Collateral compensating circulatory passages commence to develop from the first and progress to an extent that is embarrassing to the surgeon. But although this compensating circulation is sufficient to maintain life and moderate function in the limb, it is not sufficient to prevent the patient from falling into more and more depressing invalidity and from becoming unable to meet the needs of normal economic life. Such a patient, examined by Leriche six years after the start of the illness, is described thus: "His legs were enormous and a hard œdema of bluish tinge extended up well on to the abdomen. The venous network of the abdominal wall was visible and there were huge varices on both legs and two painful ulcers . . . on the left leg. Surgical exploration showed the left iliac artery to be lying free from the parietes almost against the peritoneum, and beneath it was a dense inflammatory mass of glands traversed by many small veins and enclosing the iliac vein so closely that neither it nor the lumbar sympathetic chain could be dissected free. The abdomen had to be closed with nothing useful achieved."

This knowledge of the distressing future ahead of patients suffering from thrombosis in the *vena cava*, and of the immense difficulties that will be met with in attempting to give them relief by surgical means at a later stage of their illness, caused Leriche to determine that on the first opportunity he would undertake radical treatment early. The opportunity duly arose and the case is reported in brief. The result shows that such early radical surgical treatment can avert the dark prognosis.

The patient was a woman of thirty-five years who twenty-seven days before the operation had had a confinement normal in every way. Right iliac thrombosis appeared almost at once and seven days later there was evidence that the *vena cava* was involved. The right femoral vein on exploration contained only stagnant blood, under no pressure and with no evidence of coagulation. A segment 4·0 centimetres long was removed for histological study. The *vena cava* was then approached by the subperitoneal route. It was surrounded by a mass of œdematous and inflammatory tissue and numerous enlarged lymphatic glands. When opened its contents were fibrinous debris which adhered to the vessel wall and could not be evacuated. With considerable difficulty it was dissected free for sufficient distance to allow of the resection between ligatures of some 4·0 centimetres of the vein. Nothing further was attempted. The results of this intervention were astonishing. The temperature fell to normal in two days. The œdema of the lower limbs shrank rapidly, the circumference of the right thigh diminishing by 12·5 centimetres in eighteen days. The patient left the hospital on the twenty-first day, and when seen three months later showed neither pain nor œdema anywhere about her.

Why the procedure carried out should produce this result is difficult to explain. It was limited to the removal of small sections of the right femoral vein and the *vena cava*. Nothing was done on the left side, yet the left side shared equally with right in the benefits obtained. Nothing in the operation was calculated to restore the permeability of the venous channels. Leriche confesses himself to be unable to explain it, yet, as he says, there was no doubt about the result. Shrinkage of the left thigh became obvious within fifteen hours of the operation and continued unabated. He states that in previous thrombectomies he had already noticed a rapid and bilateral disappearance of œdema after an entirely unilateral resection without clearing of the venous channels. The case reported here is only a more striking instance of the same phenomenon. To attribute it to sympathetic action is only to play with words, although it is attractive to do so, knowing how well in line with the effects of sympathectomy such a result is. The case is reported as showing a brilliant success from an operation which fell far short of what he felt might have been accomplished.

ARTHUR E. BROWN.

VASODILATATION BY ARTERIAL PERFUSION.

Jacques Oudot: *"Une méthode de vasodilatation active (applications chirurgicales)"*, *Journal de chirurgie*, Volume lxiii, 1947, page 279.

THE surgical production of vasodilatation by such negative methods as suppressing sympathetic stimuli is a well-established procedure. But the production of the same effect by direct stimulation of the parasympathetic nerves is still novel. It has been performed by Oudot by means of intraarterial injections, whereby the effect is directed locally to the peripheral nerve endings of the part selected, and the general effects of the substance injected are minimized. The possibilities and results of such a method were fully studied in a series of experiments on the isolated paw of a number of dogs, and the results of these experiments are recorded in the early part of this paper. They were such as to encourage an extension of the principle to human patients suffering from various forms of disease resulting from arterial occlusion.

The formula used for the injections was: acetylcholine 20·0 centigrammes, potassium chloride 20·0 centigrammes, sodium bicarbonate 20·0 centigrammes, and isotonic glucose serum (at 50 per 1,000) 200 centigrammes. When this solution is injected into the femoral artery an erythema appears in one to five minutes in the upper part of the thigh and the lower part of the abdomen corresponding to the area supplied by the epigastric and circumflex iliac arteries. Gradually this spreads over the whole limb, being followed by "goose flesh" and some sweating. The patient feels a sense of warmth in the limb, which reaches the knee in five minutes and the heel in about twenty. The warmth can be felt by the hand and measured instrumentally, when a rise of 3° C. may be reached. During this stage, which lasts about half an hour, there is very little change observable in the pulse or blood pressure.

During the next twenty-four hours the patient presents in the limb treated a reaction generally indicative of vaso-dilatation. In very marked cases, by means of the "reflex of Brown-Séquard-Tholozan", a similar effect seems to be produced in the other limb. This reaction generally lasts for about twenty-four hours.

If the injection is made into the brachial artery the reactions are usually more marked. There is gross local hyperthermia and much sweating. It is common in these conditions to find a slight modification of the systemic blood pressure, a slight fall being followed by a rise. This is due to an unduly rapid diffusion of the acetylcholine and is often accompanied by a feeling of malaise on the patient's part. Any such symptoms are an indication for slowing down the rate of injection. Injections into the carotid give still more strongly marked symptoms. There is a hemifacial redness, accompanied by profuse salivation, causing uncomfortable swallowing movements. The optic fundus shows a vasodilatation and an increase in the tension of the retinal artery. There is a general tendency in patients subjected to this procedure towards an increase in the systemic blood pressure followed by a slight compensatory fall. The injection, if given at all, should be given slowly and carefully.

The main clinical interest in the method lies in its application to vascular diseases of the lower limbs, in which any general reaction is very slight, but the local effect is well defined. No accident of any consequence has occurred in the 500 injections Oudot has performed. Small hæmatomata at the injection site may be a nuisance for future injections, and some susceptible patients have suffered a transitory diarrhœa on the day following the injection, but that is all. The main danger seems to lie in the possibility of injecting a vein instead of an artery, in which case serious symptoms, such as laryngospasm, cough and dyspnœa, make an early appearance and the injection must be instantly stopped. In applying the technique to human pathological conditions it is essential for success that it should be used before the pathological

changes become irreversible, and also that the arterial tree should either still be to some extent permeable or that a collateral network sufficient to carry the injection fluid should already exist.

The patients treated included five suffering from embolism, in which the value of the method is open to question. In the first few hours, if the patient's condition is good enough, an embolectomy should be performed and acetylcholine injected into the artery at the operation site afterwards. Should embolectomy fail, the segment of artery should be resected and an injection made into each cut end of the vessel. After ten hours have elapsed and embolectomy is no longer feasible, a perfusion of acetylcholine may improve the local position sufficiently for an arteriectomy to be carried out three or four days later. The method has proved of value in cases of traumatic, infective or senile thrombosis, in which conditions also it is necessary that action should be taken early. The following case report indicates its value.

A man, aged seventy-four years, was seen three days after the onset of a sudden painful thrombosis in the right leg. The right foot was cold and cyanosed, with an erythematous zone in its external part. He was in dreadful pain, any pressure on the leg being excruciatingly painful. There was no tibial pulse and the oscillograph failed to reveal any movement in the calf. Above the knee all was normal. Previous treatment had included a lumbar injection of "Novocain", 20·0 cubic centimetres of "Novocain" into the femoral artery, 10 centigrammes of papaverine and 0·20 centigramme of acetylcholine subcutaneously, all without effect, and he was demanding immediate amputation of the leg. He was given an intraarterial perfusion, which produced a strong reaction. Three hours later the whole leg except the toes was warm and comfortable. Next day the whole limb was warm. He received as a precaution two more intraarterial injections. Eighteen days from the first injection he walked, though his tibial pulse was still imperceptible, and eleven days later he left the hospital healed.

Suggestions that can be drawn from experience in such cases are that surgical attack to repair traumatic arterial damage or aneurysms might be freed of some of their risks if they were combined with intraarterial injections of this type.

Five patients were treated for late sequelæ of frostbite. Pain was relieved with remarkable speed, trophic changes regressed slowly, ulcers healed, and mobility increased. These good results are probably due to the fact that the arterial system is less badly damaged in these cases than in other diseases. Prior to practical experience it had been feared that the vasodilatation produced might cause an increase in the existing œdema, but the actual effect was the opposite. The dilatation extended to the arterioles and the vicious circle causing capillary stasis was broken. These patients have so far given the best results on treatment by perfusion. Post-phlebitic œdema has also responded well.

Twenty-one patients suffering from senile arteritis were included in the series, and thirteen of them had gangrenous processes already evident. The immediate effect of the injection was the relief of pain, followed after an hour or two by a feeling of general well-being and an ability to extend the leg fully. The good effects achieved persisted in a sufficient number of patients to be regarded as satisfactory. Patients in good bodily condition and showing early or no signs of gangrene did best.

Of the 21 cases in this group, eight had to be classed as failures, the patients either succumbing to their disease or requiring amputation. The other 13 patients were greatly benefited. Intermittent claudication disappeared almost entirely, pain was much eased, gangrene receded, and the limb became a functional asset. In nine cases, for one reason or another, the acetylcholine injections were supplemented later by sympathectomy, either in the hope of consolidating the results gained or in bilateral cases in which the artery on one side was not injectable. It is a reasonable procedure to combine the two methods of treatment, and in fact sympathectomy should be regarded as a normal follow-up procedure after a good injection result. When diabetes is the underlying cause of gangrene the immediate results have been similar to those of senile arteritis, but relapse is to be expected more commonly. In two out of four cases of diabetic gangrene in Oudot's series relapse occurred.

ARTHUR E. BROWN.

ARTERIOGRAPHY WITH AUTOMATIC CONTROL.

L. Christophe and D. Honoré: *"L'arteriographie par injection et prise de clichés automatiques"*, *Journal de chirurgie*, Volume lxiii, 1947, page 1.

AFTER a period in which the invention of arteriography by Egaz Moniz and the magnificent work following it of the Lisbon school, particularly by Reynaldo dos Santos, had to face a very unenthusiastic reception by the medical world, a stage has now been reached in which there is a danger of the method being used without discrimination. The present authors wish to emphasize the fact that unless the

conditions under which arteriographs are taken are rigidly and accurately standardized, interpretation of the films must be faulty and misleading. In cerebral arteriography particularly the human factor of error will make a variation of a half to one second in the moment of exposure; and any two films will practically never represent the exact same stage of the injection. This explains many of the difficulties of properly evaluating arteriographs.

Christophe and Honoré present an apparatus of their own devising, by means of which the pressing of a single electric button ensures a steady injection of the contrast fluid at the desired rate and a radiographic film taken precisely at a previously decided stage in the injection. For the contrast fluid they have abandoned the use of "Thorotrast". Three reasons are given for this. The first is that in several cases autopsy has shown microscopic hæmorrhages in the brain which were apparently due to the "Thorotrast". The second is that the radioactivity of "Thorotrast" might possibly give rise later on to sarcomatous formations. The third is that as all the factories producing "Thorotrast" have since the war ended been in the Russian zone of Germany, it is unobtainable. On the advice of Olivecrona they turned to "Umbradil", which they find fulfils all the requirements. The design of the apparatus is so flexible that it will deliever 3·0 cubic centimetres in ten seconds or 10·0 cubic centimetres in one second, and this flexibility permits the use of the method in the arterial systems of all the areas of the body. Aortographs have proved extremely instructive, and it is possible to get films of the whole leg from the thigh to the feet by three successive exposures made with the same tube and during the same injection. Arteriography has in fact been standardized, and with the use of an apparatus such as this bids fair to become a method of routine investigation.

The apparatus consists of an injecting mechanism and a mechanism for automatic control of the exposure. An electric motor with variable speeds propels by means of a toothed rack a metal rod, one of whose ends abuts on the plunger of a thirty cubic centimetre syringe. A speed regulator controls the speed of the injection, while a small lever disengages the rack when required. Finally, a magnet is added, which by its action on a piece of soft iron in the end of the propelling rod, disengages the rack at the end of the movement and prevents any reflux into the syringe.

The automatic release for the X-ray tube is composed of a slide, mounted parallel to the propelling bar, and carrying two platinum stops separated by a small spring. At the moment of functioning a blade comes in contact with this slide and closes the gap between the stops, thus completing a circuit through an electromagnet which activates the X-ray machine.

The whole apparatus is mounted on a jointed arm, which can be fixed to the edge of the X-ray table and moved across it close to the patient.

ARTHUR E. BROWN.

Reviews.

The Causation and Treatment of Delayed Union in Fractures of the Long Bone. By KENNETH W. STARR, O.B.E., F.R.C.S., F.R.A.C.S.; 1947. London: Butterworth and Company, Limited. 8½″ × 6½″, pp. 248, with 106 illustrations. Price: 53s. 6d.

THIS prize-winning Jacksonian essay is a masterly contribution to the surgical literature of this difficult subject. In his approach to this problem the author has first considered the morphogenesis of bone. He has delved into the history of this obscure subject and has presented the facts of our present conception of ordinary bone formation in a very clear, concise fashion. From this the next logical step has been to survey the surgical pathology of bone formation taking place at the fracture site. Here again the author has made an historical survey of this subject. He, however, has been impressed by the lack of attention that has been paid by previous writers to the role of bone resorption in the reconstitution of bone after fractures. In fact he is of the opinion that this is the key to bone formation in fractures. This view is novel, but the author puts forward some very convincing arguments backed by evidence from experimental work on animals and also from evidence gained from his own treatment of fractures. In effect he states that bone resorption precedes callus formation. The causes of non-union listed by the author on page 110 are those generally accepted.

The author holds that in the past decade the whole of surgical thought has been directed towards the immobilization of fractures by means of external splinting. He stresses that the time has now arrived when the surgeon should be bold in attacking these fractures immediately and in securing accurate anatomical position by means of internal fixation under cover of chemotherapy.

The findings of the British Medical Association subcommittee on the treatment of fractures are quoted. The best functional results were obtained from the best anatomical reduction of fractures.

The author advocates the much more general approach to the difficult fracture by means of open operation and stresses that with the compound fracture which is difficult to reduce or to maintain in reduction there should be no compunction on the part of the surgeon in immediately fixing the fractures by some means of internal fixation, plating being the method of choice. He quotes a series of compound fractures due to battle casualties treated by this method. Very few surgeons in civilian practice will have had the opportunity of treating such numbers of fractures by this method. The advocacy of this treatment is likely to give rise to a good deal of controversy amongst surgeons, especially of the older school. There is no doubt that the advent of the "sulfa" group of drugs and penicillin has opened a new era in the surgical treatment of these conditions.

Attention is drawn to the fact that delayed union is apt to occur at the sites of certain bones no matter how adequate the immobilization has been or how perfect the reduction of fracture. The sites at which delayed union is most commonly encountered are tabulated on page 116. They have these features in common: poor blood supply and dense compact diaphyseal bone.

The process of bone resorption will of necessity be slowed. To aid in the healing of the fracture the author invokes the aid of intermittant venous congestion. This was used by Hugh Owen Thomas, who also added hammering to the damming. This venous congestion is followed by a reactive hyperæmia.

The author stresses the fact that each fracture must be considered separately and all factors assessed which might delay the union of the fracture.

The new view put forward by the author, however, is that the failure of bone regeneration is due to failure of bone resorption at the fracture ends and that this is likely to occur in the dense, compact bone, particularly of the diaphysis. In order to stimulate bone regeneration he considers that bone resorption must first take place and in order to further this he drills the compact bone widely each side of the fracture site. He considers that this compact bone is likely to suffer from aseptic necrosis due to loss of blood supply from stripped and torn periosteum and that the resorption process will be aided by vascular channels which are able to grow in from the granulation tissue formed in the drill holes. The stimulation of bone formation by drilling is not new, but in the past has been carried out when bone regeneration and the union of the fracture have been delayed. The author, however, would short cut this delay in resorption and advocates immediate drilling of the compact bone, which he considers is likely to be deprived of blood supply. Such drilling is carried out in both open and closed fractures. Cortical bone grafts are likewise drilled.

The idea is novel and the author advances convincing arguments for the adoption of this method. Vigorous and scientific use must be made of chemotherapy to prevent and combat any sepsis. Where there has been any skin loss, adequate skin covering must be provided by plastic procedures. In all other aspects, however, his method of treatment follows more or less traditional lines.

The author sums up: "The essay advocates a new attitude to the pathological problem of delay in union of fractures. It is necessary that the traumatic surgeon should abandon the mechanical for the biological and laudable pus for scientific chemotherapy. The future objectives should therefore become: (1) the prevention of delayed union by effective primary treatment based on a more comprehensive surgical pathology, (2) the elimination of purulent discharge."

The book is admirably produced, well illustrated and easy to read. Short histories of fifty cases are given in the appendix. A most exhaustive and complete survey of the whole of the literature on bone formation is given. It is a most stimulating and provocative book and will be widely discussed in all surgical circles, and should do much to solve this difficult problem, which has never been adequately and comprehensively dealt with previously in the surgical literature.

Modern Plastic Surgical Prosthetics. By ADOLPH M. BROWN, M.A., M.D.; 1947. London: William Heinemann (Medical Books), Limited. 9″ × 5¾″, pp. 304, with 277 illustrations. Price: 35s. (Sterling) net.

THERE has long been a need for a comprehensive work on modern prosthetics and its association with surgery. In this book by Dr. Adolph M. Brown there is a wealth of technical detail with a mine of information concerning the development and processing of the increasing variety of substances used in this work.

However, it is perhaps unfortunate that at the outset the author states that the book is written for the general practitioner, for the value of the book lies more in its description of technical methods than in its attempts to set out the relationship between

modern prothetics and plastic surgery. One cannot help feeling that perhaps undue stress has been laid on some of the less important prosthetic appliances at the expense of others more closely related to surgery and of more importance to the patient's well-being. For example, there is little or no mention of the wide field of intraoral prosthesis and too little attention is paid to function in the discussion of amputation prosthesis for the upper limb. Many would quarrel too with the widespread use of buried implants, although none would deny their occasional value.

The problem of where surgery ends and prosthesis begins is always difficult, but one must constantly be aware of the limitations of surgery in certain cases. This book will form a valuable addition to the library of both the surgeon and the prosthetist, and will be of interest to many who are unaware of the increasing scope of this work.

La chirurgie biliaire, sous controle manométrique et radiologique peropératoire. By PIERRE MALLET-GUY, RENE JEANJEAN and PIERRE MARION; 1947. Paris: Masson et Cie. 12½" × 10", pp. 140, with 140 figures.

CLINICAL investigation and present methods of radiography are sharply limited in the information they supply to the surgeon. In particular they leave a wide gap in knowledge of the condition and tonus of the biliary passages and of the sphincter of Oddi. The authors have devised a method of obtaining manometric readings of biliary pressure correlated with radiography which, after an experience of 500 cases, they believe will provide the missing information.

By means of a cannula, a T-tube, a syringe and a manometer they are able to take tracings of the intraductal pressure after an injection of sterile water. The immediate substitution of contrast fluid for the water enables them to relate this reading to a film. The apparatus is described in detail in the first chapter of the book. The authors claim that their method will clarify many problems of biliary surgery in the same way as aerial reconnaissance will clarify the chaotic picture of a battlefield. But they admit that the advent of the aeroplane has not done away with the need for infantry patrols.

There is little doubt that Mallet-Guy and his confrères have put up a good case for the value of their technique. Cholangiography of course is no new thing, but the particular manner in which these authors use it will undoubtedly increase the ability of surgeons and radiographers to interpret anomalies in the ducts beyond the limits of simple dilatation, and knowledge of the physiology and pathology of the biliary system should benefit. The authors' conceptions of hypertonia and hypotonia of the various parts of the system will bring to most readers a sense of something in the nature of new fields to explore. Certainly their manometric tracings and radiograms taken before and after the application of the selected treatment (in the main trans-duodenal sphincterotomy for hypertonia and splanchnic section for hypotonia) enhance their case. The reported histological changes in the duct walls are not documented, as are the other aspects. In respect to obstruction to bile flow from other more mechanical causes their views have less novelty, although the motif of hypertonia and hypotonia runs all through them. But their technique will have a certain value none the less.

Altogether the book is a first-class record of an excellent piece of work. No one need be deterred from consulting it by reason of lack of knowledge of the French tongue. The illustrations, profusely scattered, will very largely tell their own story. It is, however, a dark mystery, only intelligible to the mind of the French publishing trade, that there should have been issued from a house of Masson's reputation a book of such importance, so beautifully printed and illustrated on such first-quality paper, but with its edges uncut, apparently sewn by a child, and with the covers falling off before it even reached our hands.

Proceedings of the Royal Australasian College of Surgeons.

THE GORDON CRAIG LIBRARY.

THE following is a supplementary classified list of material added to the Gordon Craig Library since the dates on which the catalogue was published and since the announcement made in the January, 1948, issue of the journal.

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H. G. WHEELER,
Secretary.

ROYAL AUSTRALASIAN COLLEGE OF SURGEONS.

Balance Sheet as at January 31, 1948.

LIABILITIES.	£ s. d.	£ s. d.
General Funds—		
Examination Expenses Reserve Account		573 19 4
Investment Fluctuation Reserve Account		83 13 0
Surplus Account		15,208 19 0
		15,866 11 4
Sundry Creditors	59 12 3	
Examination Fees Paid in Advance	68 5 0	
Post-Graduate Fees Paid in Advance	157 10 0	
Subscriptions Paid in Advance	147 0 0	
		432 7 3
		16,298 18 7
Trust Funds—		
T. F. Ryan Endowment Fund	£5,100 0 0	
Michael Ryan Scholarship Income Account	124 13 0	
J. P. Ryan Scholarship Income Account	121 5 9	
	5,345 18 9	
Syme Memorial Endowment Fund	2,850 0 0	
Syme Memorial Endowment Income Account	235 16 11	
	3,085 16 11	
Herbert Moran Memorial Endowment Fund	1,000 0 0	
Herbert Moran Endowment Income Account	26 0 8	
	1,026 0 8	
Rupert Downes Memorial Endowment	640 0 0	
Less Endowment Income Account in Debit	1 3	
	639 18 9	
Archibald Watson Memorial Endowment	896 9 2	
Less Endowment Income Account in Debit	1 3	
	896 7 11	
Craig Endowment Fund, as per Separate Balance Sheet	71,188 12 3	
		82,182 15 3
		£98,481 13 10

ASSETS.	£ s. d.	£ s. d.
Account General Funds—		
Great Mace at Valuation		500 0 0
The Union Bank of Australia, Limited. Balances at Credit—		
Melbourne	1,091 8 10	
Wellington	139 12 2	
	1,231 1 0	
Cash on Hand	20 0 0	
Advances to Honorary Secretaries	140 11 4	
Sundry Debtors	59 18 7	
Subscriptions in Arrear	21 0 0	
		1,472 10 11
General Investments		14,326 7 8
		16,298 18 7
Account Trust Funds—		
Investments—		
T. F. Ryan Endowment	5,345 18 9	
Syme Memorial Endowment	3,085 16 11	
Herbert Moran Memorial Endowment	1,026 0 8	
Rupert Downes Memorial Endowment	639 18 9	
Archibald Watson Memorial Endowment	896 7 11	
Craig Endowment Assets less Liability (as per Separate Balance Sheet)	71,188 12 3	
		82,182 15 3
Building, Furniture and Fittings (written off against Surplus)		Nil
		£98,481 13 10

Audited and found correct.
(Signed) YOUNG & OUTHWAITE.
Chartered Accountants (Aust.),
368 Collins Street, Melbourne.
March 23, 1948.

(Signed)) F. GORDON BELL, President.
(Signed) BALCOMBE QUICK, Honorary Treasurer.
(Signed) H. G. WHEELER, Secretary.

ROYAL AUSTRALASIAN COLLEGE OF SURGEONS.

CRAIG ENDOWMENT.

Balance Sheet as at January 31, 1948.

LIABILITIES.	£ s. d.	£ s. d.
Endowment	55,169 17 6	
Investment Fluctuation Reserve	141 12 10	
		55,311 10 4
Scholarship Fund		7,684 0 7
Surplus Account		8,193 1 4
Sundry Creditors		62 1 0
		£71,250 13 3

ASSETS.	£ s. d.	£ s. d.
Account Endowment—Investments		55,311 10 4
Account Scholarship Fund—		
Investments	6,650 0 0	
The Union Bank of Australia, Limited, Current Account	1,034 0 7	
		7,684 0 7
Account Surplus and Creditors—		
The Union Bank of Australia, Limited, Income Current Account	1,261 19 5	
Gordon Craig Library at Cost	6,992 7 2	
Sundry Debtors	15 9	
		8,255 2 4
Furniture and Fittings (written off against Surplus)		Nil
		£71,250 13 3

Audited and found correct.
(Signed) YOUNG & OUTHWAITE.
Chartered Accountants (Aust.),
368 Collins Street, Melbourne.
March 23, 1948.

(Signed)) F. GORDON BELL, President.
(Signed) BALCOMBE QUICK, Honorary Treasurer.
(Signed) H. G. WHEELER, Secretary.

Notes and Comments.

EXTIRPATION OF PAROTID TUMOURS.

With the possibility of post-operative facial palsy or recurrence of the tumour in their minds, many surgeons are dissatisfied with the usual methods of removing parotid tumours. I can recall some years ago removing a mixed tumour from the parotid of a probationer nurse, and my anxiety lest this lovely young woman's face should be disturbed in consequence. Fortunately it was not, but in this and similar cases the fact that facial palsy has not occurred must often be more by luck than skill, as the exposure is always inadequate through the incision parallel to the zygoma recommended in the textbooks of operative surgery.

One of the essentials of good surgery is adequate exposure, and in the case of the parotid this is possible through the incision advocated by Hamilton Bailey (*British Medical Journal*, Volume i, 1947, page 404) and now practised by him in operating on some sixty odd patients with parotid tumour. I recently had an opportunity of seeing him operating on one of these patients and was impressed with the advantages of the exposure. A long incision is made from over the base of the mastoid, well behind the pinna, downwards and forwards across the neck and along the anterior border of the sternomastoid muscle. This incision is connected in its upper part with another curved incision which begins over the parotid in front of the pinna and extends downwards and backwards. The sterno-mastoid muscle is retracted, the external carotid artery is ligated and the mastoid process is chipped off with a chisel or osteotome, the stylomandibular ligament is divided and the parotid is exposed from behind and on its deep surface and the trunk of the facial nerve is sought and traced forwards. The nerve does not, as is so frequently taught, plunge into the main mass of the gland, but with its division into its two main branches, the temporo-facial and the cervico-facial, lies between the superficial and deep lobes of which the gland is composed. The two branches are separated by the isthmus which unites the lobes. The pre-auricular skin flap is dissected forwards and the superficial surface of the gland is cleared. Branches of the facial nerve leaving the anterior border of the gland are sought and dissected out. One or two of these may be damaged, but usually without appreciable effect on the patient because of the anastomotic loops which connect the main branches of the nerve. It is now comparatively easy to effect either a radical removal of the whole parotid gland or to extirpate a tumour along with surrounding parotid tissue so as to minimize the chances of recurrence; in practice this usually amounts to removal by an incision through the isthmus of the superficial lobe in which the tumour usually lies.

There can be no doubt that by the route described safer and more adequate surgery can be performed for the effective extirpation of parotid tumours.

[From a correspondent in Britain.]